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Editorial

On Watching Operations

Hardly one of us has not been guilty of saying on occasion "I just watched the great so and so in action", and have felt a sneaking feeling of accomplishment in doing so. Many of these tête-à-têtes have been from the close vicinity of a stand or behind a closed glass, where the sure movements of the surgeon's shoulders, if not his hands, can at least be seen.

There are only a few students who have the presence of mind to ask questions of the surgeon—if he does know, he will be only too glad to answer; but in the presence of the chief, the senior interne, the junior interne, the retractor holder, and a couple of operating room nurses, one hesitates to be thus crass. And so the operation passes; we fool ourselves that we are learning something. Of course something may be learned from the drama of the operating room: operating room technique, how careful each and everyone of the operating team is in avoiding infection, what emergency measures are taken when the patient is low, the art of anaesthesia. If the patient's case history is known to you, the operation is doubly interesting and doubly instructive. If you have diagnosed a stone in the Common Duct, and then see one extruded from it before your eyes, you are both more alive to the true condition, and have more faith in a future diagnosis of a case with much the same signs and symptoms. And if you see this patient go on to a speedy recovery after the operation, you will know from your own experience that removal is the best and indeed only form of treatment. But if, as it generally is, watching an

operation is an isolated procedure, you will be none the wiser for your time spent in the operating room, and you might better have employed this time, in say, the pathology musuem, reading histories, looking closely at specimens, and relating symptoms and lesions.

Almost every one of us has had ideas of being a surgeon sometime in our course, and these ideas are exploited by friends and relatives, by the "only" girl and by popular fiction. The question is often put to us "are you going to take a post-graduate course in surgery, or be just another pill roller". Truly good results in surgery are spectacular, in medicine often as good but slower. Let us be critical of results in surgery, for if we are there will be far fewer operations done, and better ones. Our best surgeons certainly do not rush in where angels fear to tread. It is enough for us, as students, to find out the reason why an operation was done, and the results accruing, and not have visions of becoming master manipulators overnight.

The Specialist and General Practitioner

How many times in our clinical years have we heard it said: "This patient was seen by his own doctor before coming to hospital and diagnosed such and such, wrongly to be sure". The specialist has many things in his favour:

1. First of all, he has the time element. By the time he sees the patient, the disease may be so well advanced that even a lowly medical student might fare none too badly. Diseases are hard to recognize in their infancy; it takes a lot of juggling of probabilities to diagnose crampy abdominal pain as appendicitis; before it shifts to the right lower quadrant, becomes steady, and is accompanied by tenderness.

2. Secondly he is surrounded by scientific facilities which are often denied to the general practitioner. Many a diagnosis has been changed on a W.B.C. count. Of course this does not excuse him, for he should know (we often wonder how many do) how to do such a simple procedure; but he may have been too busy, his counting chamber or pipette lost, strayed or stolen away.

3. And we must include that the specialist is better trained. Accustomed as he is to rigorous criticism of his confrères, and the stark realism of the autopsy table, he is more careful of his diagnosis, makes a more thorough examination, and must of necessity be more alert to the probabilities in his own particular field.

We do not smooth over gross stupidity when we put in a word for the general practitioner, but we do believe that he gets much needless criticism for mistakes so common to all of us. When you consider the long hours, and the often poor return both in thanks and in money, it is the act of an unkindly man to burden him further with a bitter reproval about his lack of knowledge. We as students are often more guilty than the specialists themselves in loudly condemning such and such a practitioner—the young are always intolerant—and if we consider that most of us will be general men, we must look forward to being frowned upon by both the young and foolish, and the old and wise.

This calls to mind though the intolerance of the older practitioners for the young man when he first comes into the community. They say: "What can this young whipper-snapper possibly know about disease, he has never practised medicine"! How much of this is professional jealousy, how much fear of their bread and butter or of their own ignorance is hard to say. Truly the young man is often unwilling to learn from the older one; in equal or greater proportion, however, is the older man unable to "take anything" from the younger one. This is extremely unfortunate for both, for each has so much to offer the other, the older man sound and mature judgment and a knowledge of humanity; the younger man new methods of examination and the latest and often best types of treatment. There does seem to be a lack of brotherhood in medicine, which we should all strive for. There's a great field for all of us, and for all our different talents.

LOCAL ANAESTHESIA IN REDUCTION OF FRACTURES

By J. HAROLD COUCH, M.A., F.R.C.S.
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Fractures are notoriously painful injuries. The examination, transportation and treatment causes the patient extreme, and useless, suffering unless something be done to protect them. In fact the reflex spasm of muscles set up by the slightest movement, is one of the main causes of further displacement, and one of the greatest factors militating against easy and accurate reduction. Therefore, the sooner pain is relieved the sooner will the patient be protected from unnecessary suffering and the sooner will the tissue be protected from damage.

Relief from pain may be accomplished by any form of anaesthesia. In some cases it seems advisable to use general inhalation anaesthetic, in some a spinal anaesthetic and in some, a local infiltration. This last, namely local anaesthetic, is becoming rapidly more popular and is being found so satisfactory that the student would be well advised to familiarize himself with the technique before severing his association with a teaching hospital.

Technique

The actual administration of local anaesthetic in recent fractures is more simple than in any other type of surgery, but it must be carried out under the same aseptic technique as for any open operation on bone. The skin is prepared for operation using whatever routine one has adopted. We use gasoline, ether, iodine, alcohol. Sterile towels are draped in position around the area. The surgeon wears sterile gown, gloves, a cap and mask.

Actual site of fracture is found by palpating along the shaft of bone until a point of local tenderness is located. A skin weal is raised with novocain and through it the surgeon then proceeds to "needle the bone" using a long fine needle on a syringe, until the needle drops over the fractured end of bone. If point of needle is in correct place between fractured ends, it should also be in the haematoma which gathers at fracture, and correct position can be proven by withdrawing a small amount of blood into the syringe. If no blood can be withdrawn, needle is not in right place. When accurate position has been proven, 20 c.c. of 2 per cent novocain solution is injected into the haematoma whence it spreads to the tissues around the broken ends and produces rapid relief of pain and relaxation of muscle.

Fluoroscopic screen, when available, may also be used to make sure the point of the needle is in the correct place. The unsterile screen must not be placed directly on the skin but the surgeon's hand, syringe with needle, and site of fracture may all be covered with sterile towel and the screen placed on top of the towel permitting point of needle to be accurately placed under direct vision.

Colles' and Pott's Fractures

Unfortunately the two most common fractures are the two most difficult to treat under local anaesthetic and disappointment is sometimes expressed by those making the attempt and finding it unsatisfactory. If, however, reason for failure is made quite clear, it is possible to handle a Colles' or Pott's fracture just as satisfactorily as any other fracture.

The essential point to be remembered is that a Colles' fracture or a Pott's fracture is a double fracture. If two bones are broken, two bones must be anaesthetized through two separate points of entry, using two quantities of fluid.

In a Colles' fracture, the end of the radius and ulnar styloid must be anaesthetized separately. Sometimes in this particular fracture, impaction is so great and haematoma is so small that the needle must be insinuated between the fracture ends from both dorsal and

ventral aspects to get solution in contact with fracture and so produce anaesthesia. (See Figs. 1a, 1b.)

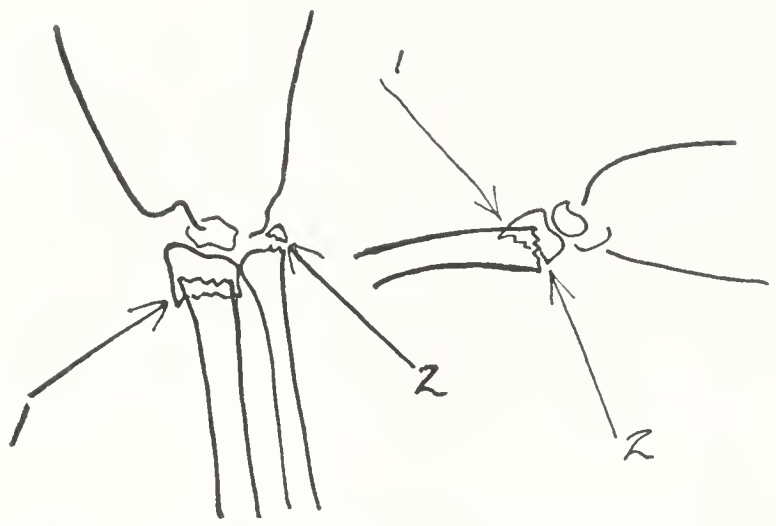

Fig. 1 (*a*).—Colles' fracture. Radius and Ulna both broken therefore both must be anaesthetized.

Fig. 1 (*b*).—Colles' Fracture. Impaction sometimes so great and haematoma so small that needle must be insinuated between the fragments from both front and back.

In Pott's fracture the fracture of fibula and the fracture of the medial malleolus of tibia must be anaesthetized separately. (See Fig. 2.)

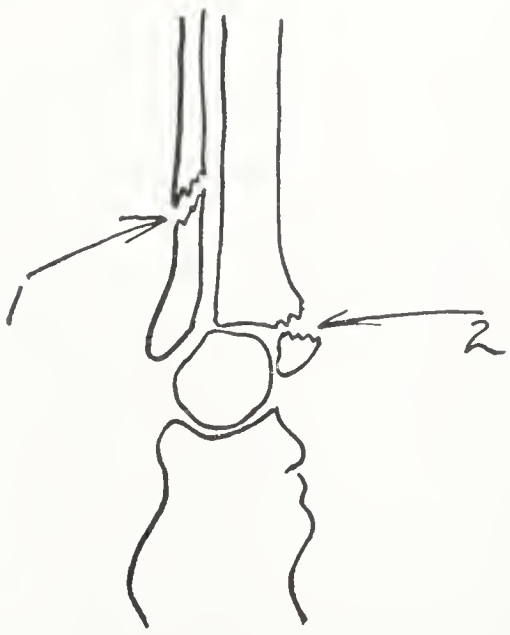

Fig. 2.—Pott's Fracture. Both fibula and tibia are broken so both must be anaesthetized.

Advantages

The advantages of local anaesthetic are so obvious that it is surprising how slow we have been to use it. Only within the past 10 years has it sprung into general favour.

In the first place, its wide applicability commends it. Fractures of almost every bone in the body can be reduced under local anaesthetic. The simplicity of the procedure places it in the hands of every doctor who is called upon to treat fractures. The administration of this type of anaesthesia, which is not much more complicated than the giving of a hyperdermic injection, is so much simpler than the use of local for operation on hernia or goitre that any surgeon who is at all accustomed to handling a syringe and needle should be setting his fractures with this aid.

Local anaesthesia avoids all the risks that are associated with general anaesthetic. There is no general inhalation anaesthetic which is absolutely safe. All have their share of complications such as pneumonia, aspiration of vomitus, collapse of lung, shock. Even the post-anaesthetic nausea and vomiting which is more distressing than dangerous is eliminated.

Fractures may be reduced without need of an anaesthetist. While we find it reassuring and helpful to have an expert anaesthetist looking after the comfort of the patient in a large hospital, there are many doctors who find themselves confronted with a fracture where no confrère can be easily called in and to such the local anaesthetic should be a real boon.

Disadvantages

If local anaesthetic is to be used satisfactorily, its few disadvantages must be recognized and taken seriously.

Theoretically the surgeon has converted a simple fracture into a compound fracture by the introduction of a needle through the outside skin thereby increasing the chance of infection. We have never seen any bad results which could be attributed to this procedure but this theoretical consideration must be mentioned to emphasize again the need for aseptic technique and complete cleanliness in using local anesthetic. If such ordinary surgical technique is observed, the risks are neglible.

In complicated fractures with open wounds which are potentially infected, local anaesthetic can be used but injection should be made through nearby healthy skin observing the same technique as before. It should not be

introduced through the wound. Danger of infection is inevitably greater in complicated fractures than in simple fractures.

Large blood vessels, joints or tendon spaces, may be filled with the injection solution instead of the haematoma between the bones. This inaccuracy may be avoided if one merely keeps in mind the anatomy of the surrounding tissues.

A very real danger is produced in the complete elimination of pain as a result of the introduction of local anaesthetic. Patients sometimes make attempts to move the broken part or even to use it, thereby producing more trauma. This danger can obviously be avoided once it is thought of by keeping close watch over the patient and by warning him to avoid unnecessary movement.

Since local anaesthesia carries with it no post-anaesthetic effects, patients are often allowed to go home immediately following the reduction of the fracture which removes them from expert supervision. If a circular plaster is used, the danger of impaired circulation, as a result of swelling under the cast, must be guarded against. If there is any likelihood of swelling following application of plaster the patient should not be trusted to the observation of himself or to untrained relatives but should be kept in hospital until swelling has occurred so that diminished circulation may be recognized. The advantage of being allowed home is not sufficient to offset the disadvantage of Volkman's ischaemic paralysis or gangrene.

On Medical Libraries

"Now there has come a great change in our time over the form in which living thought presents itself. The first printed books,—the incunabula,—were inclosed in boards of solid oak, with brazen clasps and corners; the boards by and by were replaced by pasteboard covered with calf or sheepskin; then cloth came in and took the place of leather; then the pasteboard was covered with paper instead of cloth; and at this day the quarterly, the monthly, the weekly periodical in its flimsy unsupported dress of paper, and the daily journal, naked as it came from the womb of the press, hold the larger part of the fresh reading we live upon. We must have the latest thought in its latest expression; the page must be newly turned like the morning bannock; the pamphlet must be newly opened like the ante-prandial oyster. Thus a library, to meet the need of our time, must take and must spread out in a convenient form, a great array of periodicals."

Oliver Wendell Holmes,
Dedicatory address at the opening of
the Medical Library in Boston,
December 3, 1878.
The Riverside Press, Cambridge.

ANAESTHESIA

By G. RUPERT BALFOUR, B.A., M.D.
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Anaesthesia is now undergoing a profound and significant change. It is, at last, definitely and finally passing into the hands of the anaesthetist. Recent anaesthetic practice has had two main characteristics. The first is that the introduction of new drugs and new methods has made anaesthesia more complicated and the selection of the best anaesthetic for a specific surgical situation more difficult. The second is that many surgeons recognize the need for revaluation in anaesthesia and look to the anesthetist for expert advice as well as for administrative skill. This demand promises to break through the crust of custom and offer a chance for something better in anaesthesia.

There is nothing of finality in anaesthesia. It is rather a growing body of knowledge constantly being revised or replaced by new knowledge. Its changes have never been revolutionary, forced by some massive pressure of facts. They have come slowly and grudgingly, with much trial and error, with discord and dissolution.

Anaesthesia was born into obscurity, the younger son without inheritance, hidden in the shadow of the dramatic and vital growth of surgery. A hundred years ago ether, chloroform and nitrous oxide were introduced. There have been no important additions to these drugs until recent times. For years laboratory workers have been warning of the dangers of chloroform yet only recently has its use been curtailed. Why has this lack of interest, this apathy, towards anaesthesia existed? The anaesthetic does not enter into the conscious experience of the patient to any appreciable degree and it is accepted as an inevitable and necessary part of an operation. And people are singularly lacking in imagination. Because they have no imaginative experience of the suffering they so commonly cause, they are cruel. No more do they realize what an operation would have meant in pre-anaesthetic times. If an anaesthetic were difficult to get, rare, costly or dangerous, it would be valued differently. But the patient is reassured: there is practically no danger, no discomfort in the taking; there will be no bad after effects; the cost is very little. Constant repetition of an understatement inevitably results in its acceptance. The recognition that anaesthesia involves danger and therefore must be taken more seriously, is not to be discouraged.

There is another reason why anaesthesia has, in the past, remained in obscurity. The anaesthetic was the responsibility of the surgeon and the administrator was too frequently selected for reasons other than his skill. Ether and chloroform were used because it was thought that these drugs were safest in inexperienced hands. The development of anaesthesia as a specialty is of such recency that its importance has not been recognized.

There is much to be said for the practice of anaesthesia as a full-time specialty. Up to the present time this has not often been possible. In all probability increasing opportunity will be afforded to trained anaesthetists for staff appointments in the larger medical centres on such a basis. In smaller cities and in rural practice anaesthesia will of necessity remain linked with general practice. It will, however, remain a specialty with adequate reward for the time spent in training. The fact that surgeons of superior ability and training are going to these smaller hospitals assures the anaesthetist's position as they are most unlikely to be satisfied with an inferior standard of anaesthesia.

The anaesthetist must of necessity develop skill in the administration of the frequently used anaesthetics. At the present time 95 per cent of our anaesthetics are ether, cyclopropane or spinal. Nitrous oxide is used in not more than 4 per cent and all the others in less than 1 per cent. These figures may change.

New drugs, new methods, may appear to be mastered. But it is not technical skill alone which will mark the superior anaesthetist. He will be the one best able to assess the risk, to judge wisely, to choose the right anaesthetic, taking all factors into consideration.

The choice of anaesthetic has through custom been the duty of the surgeon. This is reasonable in that he has examined the patient and understands the surgical problem involved. From his experience he has formed an opinion of the value of the various anaesthetics used. This opinion has unfortunately often become a fixed preference which has led to an arbitrary valuation, dogmatic and inflexible. We talk about one surgeon preferring to operate with spinal, another preferring ether or cyclopropane and there is the tendency to fit the patient to the anaesthetic. Every anaesthetist, on the other hand, is aware of the great difference in the way patients respond to the same anaesthetic technique. That there is no easy and simple solution to the problem of anaesthetic choice and that each patient must be considered as a separate problem is now evident.

To determine with certainty the anaesthetic proceedure best suited to meet a particular situation is most difficult. Essentially it is the method which involves the least risk to the patient. Risk is a complex consideration. It implies a voluntary acceptance of danger, a choice, the weighing of this against that, the possibility of failure. The simple enumeration of deaths per thousand anaesthetics is as valueless as an estimate of the behaviour of the average man in predicting an individual's next move. We will continue to use anaesthetics of higher death rate in operations of higher mortality to save the lives of patients to whom these more risky procedures offer greater chance of recovery than do statistically safer methods. More patients in advanced years with intestinal obstruction or cancer of the stomach are alive because a better operation was done with a more dangerous anaesthetic. But when the choice is made, the odds considered, the risk accepted, a failure must not be permitted to discredit the wisdom of the selection made.

How shall a proper determination of risk be made? Risk must be, to use a modern term, a total risk, the summation of all component risks, the interrelated variables independently and jointly assessed. The variables in such a conception of risk are many: the age and build of the patient, the nature of the operation, complicating pathological conditions, the risk inherent in the administration of the anaesthetic, the skill and equipment of the anaesthetist, the ways of the surgeon.

The first consideration is that of age and build of the patient. The small bodies of infants and young children tolerate ether well, but because of their low respiratory effort, do not do as well with closed circuit methods. Spinal anaesthesia is used rarely because of the unfavourable mental attitude of children to this type of anaesthetic. Older children may be given cyclopropane but the technical administrative difficulties are greater than in adults. Early adult life is without doubt the period of anaesthetic safety and in this group it is reasonable to believe that spinal anaesthesia has no greater inherent risk than has cyclopropane or ether. Spinal anaesthesia can be used with equally good results in patients of any build and the same is true of ether. But it is with cyclopropane that the difference in build becomes important. Abdominal relaxation is easily produced in the asthenic patient but the large robust muscular individual is a very different problem. Inexperienced administrators do not appreciate this fact. In such cases spinal or ether might be employed with more satisfactory results. The degenerative diseases as they appear in later adult life add definitely to the risk of spinal and this increase in risk is doubled, increased ten times, then a hundred times as cardio-vascular degeneration increases with age. Oxygen want becomes an ever present danger and the safety of ether anaesthesia is directly in relation to the adequacy with which oxygen supply to the tissues is maintained. This is probably one reason why old people tolerate cyclopropane anaes-

thesia so well, because the oxygen supply is always adequate.

The nature of the operation makes specific demands upon the anaesthetic, particularly if the abdomen is to be entered. The primary requisite is relaxation and this must be of sufficient duration for the completion of the operation. Spinal anaesthesia is without question the best means of providing complete relaxation and the patient benefits in that less difficulty is experienced in operative dissection and there is less trauma from manipulation and retraction. It is possible on some occasions to do a more extensive operation under spinal anaesthesia than would be otherwise possible such as in the case of a high-lying gastric ulcer, in gastric cancer or in the removal of calculi from the common bile duct which might otherwise be missed. The relaxation of spinal anaesthesia is practically indispensable for successful operation in intestinal obstruction when the abdomen must be closed in spite of greatly distended bowel. When for other reasons, spinal is not advisable, ether may produce comparable results with the added advantage of unlimited time. The large endotracheal catheters introduced with a laryngoscope make possible better controlled ether anaesthesia, a quieter abdomen through less respiratory effort and better oxygenation. Cyclopropane is most satisfactory in lower abdominal surgery where such complete relaxation is unnecessary. When the use of cyclopropane is imperative for other reasons the endotracheal method produces the most satisfactory results.

Nowhere in the field of anaesthesia is there greater need for revaluation than in our conception of the effect of complicating pathological conditions present before operation and in our attitude toward the post-anaesthetic complications. It is difficult with the knowledge at hand to make any definite assertions. There is good reason for not using ether in diabetics or in those suffering from nephritis. The ban on ether in chest disease is not by any means justified. While we have no definite proof as yet, it appears that chest complications following the use of endotracheal ether are less common than with spinal anaesthesia. The minimum pulmonary disturbance is caused by cyclopropane; the maximum danger is apparently in the combination of spinal and ether. We do not consider the use of spinal anaesthesia wise in the presence of spinal disease, not because of any anticipated effect of the spinal on the disease but rather because any further development of the disease may, in the mind of the patient, be linked with the anaesthetic. The safety of spinal anaesthesia is greatly increased by the pre-operative administration of intravenous saline or by transfusion when there has been loss of blood or when shock is present. Hypertension in itself is not necessarily dangerous but the patient who has previously had a high blood pressure and appears in the operating room with a greatly lowered pressure, must be viewed with apprehension, especially if there is some cyanosis present. It is the unstable, inelastic vascular system of advanced age that provides the risk in spinal out of all proportion to all other considerations. Many of the so-called post anaesthetic complications are unrelated to the anaesthetic. Retention of urine and distension are probably more closely related to surgical trauma. The responsibility for pulmonary complications must be shared equally by the operation and the anaesthetic—the frequency with which they appear following upper abdominal surgery under all types of anaesthesia suggest this. Headache following spinal anaesthetics is a real and difficult problem the answer to which is obscure. The fact that it is most commonly found following brief anaesthetics for minor operations suggests that an important factor may be the patient moving about much too soon.

The choice of anaesthetic may be limited by the skill and equipment of the anaesthetist. Ether fortunately requires no complicated and expensive apparatus and is safest in the hands of the inexperienced anaesthetist. The endotracheal technique, which has so many advantages, requires training and skill. Spinal anaesthesia is rather too

easy. Its safety is directly related to the experience of the anaesthetist, and his ability to use it with wisdom and discrimination. He must be able to detect changes in the patient's condition early and institute remedial measures before serious damage is done. Anoxia must be prevented, early evidence of shock detected and treated. Cyclopropane requires expensive equipment and so its use is limited to larger hospitals. It has turned out to be safer than was believed possible at the time of its introduction. It is now being given everywhere and by relatively unskilled anaesthetist and fatalities have been very few.

The hazard of flammability and explosibility has existed since the introduction of ether. While ether-oxygen mixtures are highly explosive as are also nitrous oxide-ether-oxygen mixtures, little attention was paid to this potential danger. More recently ethylene and cyclopropane, both of which are used with oxygen in explosive mixtures, have been responsible for a number of disastrous explosions fatal to the patient. Cyclopropane is apparently such a satisfactory anaesthetic in other ways that every attempt is being made to eliminate this risk of explosion. The only real solution for inhalation anaesthesia is, of course, the development of a non-explosive gas possessing the required anaesthetic properties. Another solution would be the use of cyclopropane combined with the other gases in a non-explosive mixture such as cyclopropane with helium and oxygen. Until this is accomplished every precaution must be taken to prevent the igniting spark, static in origin or otherwise, from igniting the mixture. For this reason the anaesthetist, patient, machine, operating table, etc., are all electrically connected and grounded. The danger is apparently, greatest from the static electricity and explosions are confined to the closed circuit of the anaesthetic. Mixtures containing less than 2 per cent cyclopropane or less than 1 per cent of oxygen do not explode, and a mixture of 2 per cent cyclopropane in the operating room is virtually an impossibility. The explosive risk is materially lowered by maintaining a high humidity. Every effort is being made to eliminate the danger but until the cause is understood we shall not be free from concern.

There remains one other factor influencing the choice of anaesthetic and that is the ways of the surgeon. Some one must consider this question of anaesthetic risk very seriously and no one is in a better position to do this than the surgeon provided that he is prepared to determine the medical condition of each patient and in association with the anaesthetist become familiar with the problems of anaesthetic administration. If he does not desire to do this the anaesthetist must make it his responsibility, attempting on his part to interpret the requirements of the operation correctly.

The future development of the specialty of anaesthesia is clearly indicated. It has lost its simplicity. The determination of the safest and best anaesthesia is in itself a matter of great importance involving the consideration of many factors. The ability through experience and judgment, to make such a decision wisely ranks in importance with the skill in administration of the anaesthetic. Such a responsibility the anaesthetist must be prepared to accept. And the assumption of this responsibility assures the future of the specialty of anaesthesia.

A PROBLEM IN DIAGNOSIS

A CASE OF ANURIA

By F. H. CÔTÉ, B.A., M.D.
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The following case report may interest you. The students who were on the ward at the time will probably remember this case. The patient died after ten days in hospital. A post mortem examination was made. At the request of the Editor, the autopsy findings are not included; they will appear in a subsequent issue of this journal.

History

A white male age 52 was admitted 10 days before death to the G. U. Service in an acutely ill condition. He was conscious, but too ill to give a satisfactory history, and most of the information was obtained from his wife.

Apparently, he had been quite well until five days before admission when he developed a sudden, severe upper abdominal pain, mainly in the epigastrium and right upper quadrant. The pain was sharp and crampy; it lasted for about six hours, and then subsided to a dull ache remaining so until admission. The patient had not been nauseated, nor had he vomited. Shortly after the onset of the acute pain, he had taken a dose of salts and soon after he began to have marked diarrhoea, which had been severe though diminishing gradually in the past five days. With the onset of the diarrhoea he passed no urine through the urethra. He thought that all the urine was being passed through the bowel with the loose stools and was unconcerned about this. His doctor, who had seen him for the first time the morning of admission, thought there was a perforation of the bladder into the rectum, and he referred him immediately to hospital.

On closer enquiry, there was a year's history of fairly typical duodenal ulcer dyspepsia with occasional passage of blood in the stools. There was no particular food intolerance. The patient was a heavy drinker, and lately he had been drinking a fair quantity of wine though not much more than usual. He had not eaten any unusual food, nor had he been taking any medicine as far as was known. He had had a lacerated wound of the right temple sutured in the Out-Patient Department of another hospital eight weeks previously. This was the only information available.

Examination

The patient was a moderately well developed, moderately obese, middle aged man. He was acutely ill. His skin was cold; he was not cyanosed; the pulse was imperceptible; the blood pressure could not be registered on the cuff. He was markedly dehydrated, fully conscious, and complained of moderately severe, upper abdominal pain. There was a recent scar over the right temple. The tongue was dry and furry; the breath had no special odour; the tonsils were not enlarged, nor the thyroid; there were no palpable lymph glands in the neck; the chest was clear. The heart was not enlarged, regular with an apex rate of 80. There were no murmurs; the heart sounds were faint. The upper part of the abdomen was moderately distended. There was some rigidity, mostly voluntary, in the right upper quadrant. There was no hyperesthesia, but there was a definite point of tenderness, maximal over the gall bladder. The kidneys were not palpable, nor tender. There was no costovertebral tenderness, no loss of liver dullness, no shifting dullness, no masses in the abdomen, the bladder was not distended. On rectal examination the sphincter tone was normal. The pro-

state was not enlarged. The vesicles, lower rectum and anal canal were normal. There was certainly no palpable perforation in either bladder or rectum. Neurological examination was normal. The extremities were normal. A catheter was passed into the bladder. There was not a drop of urine.

Laboratory Findings

Temperature 97 per rectum; Hb. 86; red blood count 4,500,000; white blood count 15,000 with 80 per cent polymorphs and 20 per cent lymphocytes. There was no urine available for examination. The NPN was 100, the Wassermann was negative.

Disposal

The shock and dehydration were treated first. The patient was put to bed with extra blankets, an electric heat cradle, and a continuous 5 per cent glucose in saline intravenous started. Coramine in 2 c.c. intravenously and intramuscularly doses alternating with caffeine sodium benzoate intramuscularly in 15 gr. doses at half hourly intervals were given until the blood pressure had risen sufficiently. A catheter was tied into the bladder, and a rectal tube inserted lest urine might be coming from the bowel. At the end of two hours the blood pressure had risen to 80/60 and in three hours was 120/80. The patient now felt warm, and looked much improved, but there was still no urine from the bladder and none from the rectal tube. The latter was then removed as it seemed fairly certain that there was not an intestinal leak of urine. During the first day the patient had no diarrhoea, and no bowel movement.

When he had sufficiently recovered from the marked cardiovascular collapse, he was examined under the screen for the possible presence of air under the diaphragm. The possibility of a perforated duodenal ulcer presented itself because of the history of dyspepsia, the occasional blood in the stools, the upper abdominal findings, and the cardiovascular collapse. It was true that he had no signs of peritonitis which one would have expected, and there was no loss of liver, or splenic dullness. With the screen, no air was seen under the diaphragm.

Four hours after admission, the patient had received some 1800 c.c.'s of intravenous fluid and still, there was no urine. It was obvious that whatever surgical condition, if any, was present in the upper abdomen, the anuria was the more serious condition requiring immediate treatment. The cause of the anuria was not clear. There was a history of wine drinking and diarrhoea suggesting a possible intoxication or poisoning.

During the first night in hospital the patient became wild and jumped out of bed. He had to be given paraldehyde. By the next morning, however, he had settled down, was quite rational, and was able to corroborate most of the details of the history given by his wife. His blood pressure was now 148/88; the tongue was slightly moist, he looked definitely improved, but during that day he passed only one ounce of urine per catheter. The urine was clear, amber coloured, acid in reaction; it contained a trace of albumin; the benzidine test was positive, and on microscopic examination there were two to five red blood cells, and three to five white blood cells. There were no casts or epithelium. The NPN which was 100 on admission had come down to 88, probably from dilution.

A search was then instituted for a possible obstruction somewhere in the urinary tract. As a catheter could be passed freely into the bladder one could rule out an obstruction from the external urethral meatus to the bladder neck.

On cystoscopic examination there was a slight, non-specific cystitis. There were no stones, tumours, nor diverticula. There were only a few drops of urine in the bladder. Both ureteric openings were normal. Ureteral catheters inserted on each side passed easily to the kidney pelves meeting no obstruction. A plain KUB (Kidney, Ureter, and Bladder) X-ray plate with the ureteral catheters in situ showed no opaque stones. The pelvis and calyces of the kidneys on each

side were normal, and the kidneys themselves were normal in size and position as far as one could tell. The ureteral catheters were tied in, in the hope that this might bring about a flow of urine as not infrequently happen with such a procedure in cases of anuria. No urine passed through the catheters, however. They were left in for one and a half days before the patient finally pulled them out himself. It was now certain that the anuria was not due to obstruction.

Meanwhile measures to promote a diuresis were being carried out. The ones used may be summarized as follows:

1. Heat to the loins; poorly tolerated by the patient.

2. Intravenous fluids; 5 and 10 per cent glucose in distilled water and in saline; 50 per cent glucose intravenously.

3. Various diuretics: mainly aminophylin up to 40 c.c. doses, repeated every four hours. Also caffeine sodium benzoate in 15-30 gr. doses.

4. Pilocarpine 1/6 gr. doses by hypo. to favour skin elimination. This was without effect, and caused a moderate fall in blood pressure.

5. Enemas and purges to eliminate possible toxins and promote fluid excretion through the bowel.

Briefly, none of the above measures were effectual in producing a satisfactory diuresis. The patient died on the tenth day. The following day by day summary may be of some interest:

1*st day:* Conscious—no pain—felt comfortable—able to eat and drink—temperature ranged from 96° to 98°—total amount of urine 1 c.c.—blood pressure 148/88—NPN 100.

2*nd day:* Restless during the first night, but conscious and rational throughout the whole day—temperature 96° to 98°—urine output 4 oz.—blood pressure 130/80—NPN 88.

3*rd day:* Conscious — general condition unchanged—temperature 97° to 98°—urine output 4 oz.—blood pressure 140/80—NPN 72. Urinalysis showed an acid urine with a specific gravity of 1018—it contained a two plus albumin —no casts, but there were 10 to 15 red blood cells, and 20 to 30 white blood cells, per high power field.

4*th day:* Conscious and rational, but slightly lethargic. Temperature 98° to 99°—blood pressure 140/85—amount of urine 4 oz. During the subsequent days the blood pressure remained fairly constant at this level, and will not be further recorded. The NPN was 160—the creatine 5.3—blood chlorides 380 mg.

5*th day:* i.e., the tenth day of illness the patient was slightly drowsy—breath not uriniferous. Urine output 17 oz.—temperature between 98° and 100.8°—respiration 25—moist rales in both lung bases—no surface oedema.

6*th to* 9*th day:* Progressively more drowsy—respirations between 20 and 30 —temperature between 98° and 99.8°—urine output 5 oz., 6 oz., 3 oz., and 4 oz., respectively, each day — patient not eating, on continuous intravenous fluids.

10*th day*: Deeply comatosed—could not be roused—breath definitely uriniferous — bilateral basal bronchopneumonia—temperature 97°—NPN 275—creatine (re-checked) 3.9—urine output 5 oz. Died.

While patient had severe diarrhoea before admission there were no loose movements while in hospital, the average being one to three fairly well formed stools a day. He vomited about twice a day in the last four days.

Observations

The following observations in this case are noteworthy:

The patient lived in all fifteen days from the onset of his illness. The longest case reported lived twenty-nine days. The total urine output during ten days was 53 oz. despite an average daily fluid intake of 2,000 to 3,000 c.c.'s. Despite this fluid imbalance there was no demonstrable pitting oedema (it is stated that for pitting oedema it is necessary to have a 10 per cent increase in tissue fluid volume). The only signs of fluid were in the chest in the latter part of the illness. There was no ascites. The liver was never below the costal margin. The clinical appearance of the patient was typical. He was fully conscious for the first five days, that is, until the tenth day of illness, then he became progressively more drowsy end-

ing in deep, uraemic coma with high NPN. He was not jaundiced at any time. All methods of treatment were notably unsuccessful. One important feature was the onset simulating an upper abdominal condition, or a gastro-intestinal upset. There was no definite clue as to the cause of the anuria. The only significant findings were drinking and diarrhoea. The stomach contents were examined on the second day, but unfortunately a chemical examination of either gastric content or stool was not made. It was reasonably clear that the anuria was not due to obstruction in the urinary tract.

Summary

When the urine output ceases entirely, or perhaps amounts to 30 to 40 c.c.,s in twenty-four hours, the term anuria is used. The bladder is usually found empty on repeated catheterization. Anuria either results from a cessation of secretory activity, termed secretory anuria, or from an obstruction to the outflow of formed urine, termed obstructive anuria.

Causes

The various causes in these two groups may be summarized briefly:

A. Secretory Group.

1. Proximal to Kidney.

1. Embolism or thrombosis of the main renal vessels of both sides.
2. Reflex inhibition of one kidney by obstruction on the other side, *e.g.*, after ureteral transplantation.

2. Renal Causes.

1. Cessation of secretion because of large extrarenal fluid losses as in severe haemorrhage, diarrhoea, vomiting, and advanced cardiac insufficiency.
2. Extensive, bilateral destruction of renal parenchyma, *e.g.*, renal tuberculosis, polycystic kidney, hydro. or pyonephrosis; certain forms of acute, non-suppurative nephritis and nephrosis, particularly mercurial nephrosis, under this heading comes the anuria seen in black water fever, diphtheria, cholera and yellow fever, and certain poisonings as lead, phosphorous and turpentine.
3. After nephrectomy if the other kidney is inadequate in function.

B. The Obstructive Group.

In this group there is an obstruction to the outflow of secreted urine. The commonest causes are:

1. Obstruction of ureters or kidney pelves by bilateral calculi, extensive carcinoma of the uterus, bladder, prostate, or other abdominal malignancy.
2. Calculus blocking the only functional kidney, the other one being destroyed by neoplasm or disease.

Clinical Pictures

A case of anuria may show one of the following clinical pictures:

(*a*) *A period of "tolerance"* — or asymptomatic stage in which the patient may look and feel remarkably well, but the blood chemistry steadily rises. This stage usually lasts about four to six days. There is no urine in the bladder or only very small amounts of blood tinged urine.

(*b*) *A period of "partial tolerance"*— After about four to six days of moderate comfort, the patient develops headache, anorexia, nausea and becomes apathetic; hiccoughs and muscle twitching may be troublesome.

(*c*) *The stage of "complete intolerance" or uraemia*—Unless the anuria is relieved the final picture may come on suddenly within a few hours or be gradual. The patient is comatosed and remains so until death. There may be generalized convulsions, tonic and clonic, particularly so, if there is vascular disease of glomerrulo nephritis. The blood chemistry is high.

Diagnosis

The clinical picture of anuria is so typical that the diagnosis is easy. The problem consists in determining the cause whether secretory or obstructive. The history often gives the clue. For instance the history of previous nepritis, or operation for stones, tumours,

or the passage of calculi and so forth will give much help. Certain urological methods of investigation will add much information. Some of these are as follows:

1. A flat or stereo KUB X-ray plate should rule out obstruction from calculi of the radio opaque variety, *e.g.*, calcium carbonate and oxalate, etc. False security is avoided if one remembers that all urinary calculi do not show in the X-ray plates.

2. A catheter passed into the bladder will rule out or overcome an obstruction from the external meatus to the bladder neck.

3. Cystoscopic examination will rule out any bladder cause, *e.g.*, stone, tumour blocking both ureteric orifices.

4. Ureteral catheterization will determine any block up to the kidney pelves on either side. Occasionally the passage of the catheters will initiate the flow of urine. If an obstruction is met with and can be passed, the catheters may be tied in.

Treatment

There is much doubt as to how much can be accomplished in the secretory anuria. One should try to eliminate toxins by all possible routes, by purges, enemas, stimulation of the skin, etc., and one should attempt to produce diuresis by fluids, diuretics such as digitalis, aminophylin, salyrgan, caffeine and sodium benzoate, etc. Decapsulation of the kidneys used to be done. Its value is doubtful; it is a heroic measure, and it is rarely done now.

In the anuria due to bi-chloride, purging with salines, fluids pushed moderately, and the administration of sodium thiosulphate are usually tried.

Particular attention is drawn to the occasional fatal anuria, which may follow blood transfusion. It is a most distressing complication in an otherwise satisfactory surgical case. Some five or six such cases are in record in this hospital. The reactions during or following transfusion of unsuitable blood are several. Some, for example, protein reaction, serum sickness, may not be serious. Haemoglobinuria is another complication of transfusion. It consists in the presence of blood pigment in the urine without corresponding red blood cells. It indicates prerenal haemolysis. The finding of pigment in the urine in the absence of sufficient red blood cells to account for the amount is diagnostic. The causes of transfusion reaction are many, and are not discussed here. The best treatment here, however, is prevention. Strictest attention should be paid to the minutiae of transfusion technique to insure asepsis while taking and giving of the blood, and to prevent haemolysis of the donor blood. The compatability of the donor and recipient bloods should of course always be determined before. The problem, as to why untold reactions occur during blood transfusions is complicated, and yet unsolved. It is the subject of a special study in this hospital at the moment.

In the obstructive cases of anuria treatment is directed towards any of the possible obstructive causes mentioned above, and will necessary vary from a simple bladder catheterization, ureteral catheterization to nephrostomy, ureterostomy, transplantation, etc., etc. Certainly forced fluid treatment should never be instituted in this group before relief of the obstruction.

Conclusion

1. A recent case of anuria in which the patient lived fifteen days, putting out 53 oz. in the last ten days is described.

2. Anuria in general is discussed very briefly.

3. The pathological findings are left to your speculation until their subsequent publication in collaboration with the Department of Pathology.

I am indebted to Dr. Pearse for permission to report this case.

[There was no correct or nearly correct answer to this problem, so we have been unable to give away a ticket to the Meds-at-Home.]

THE ANSWER TO THE PROBLEM PUBLISHED IN THE FIRST ISSUE

By C. BROWN, M.D.

Autopsy Report

At autopsy the signeficant findings were as follows:

Lungs and pleura:

There were fibrous adhesions in both pleura cavities. Two litres of greenish-yellow fluid was present in the right pleural cavity. Both lungs were voluminous. The upper lobe of the right lung was hard and irregular. The remainder of both lungs were crepitant. Upon opening the bronchi the right upper lobe bronchus was found to be partially occluded about 3.5 cm. from the bifurcation. The bronchial wall was 3 mm. thick, yellowish in colour and gritty to cut. The right upper lobe on section was also gritty to cut. All the bronchioles were small and their walls thickened. The lung tissues was replaced in many areas by yellow, solid tissue. At the periphery there were small areas of pneumonic consolidation. The bronchial lymph glands were enlarged and had a rubbery consistency. On section the lymphoid tissue was replaced by solid, yellow neoplasm.

Microscopically, sections through the main bronchus showed the mucous membrane to be almost entirely destroyed by ilfiltrating, atypical epithelial cells which were completely undifferentiated. The lung parenchyma adjacent to the main bronchus was fibrosed and infiltrated with lymphocytes and atypical epithelial. Some of the smaller bronchioles were filled with partially organized exudate and others with fibrous tissue infiltrated with atypical epithelial cells.

Abdomen:

The peritoneum was thickened to about 2 mm. The greater omentum was rolled up and lined in the L.U.Q. It contained many nodules which on section were yellow and shiny. The mesentery of the small bowel was replaced by a nodular mass of hard neoplasm. The transverse mesocolon and pelvic mesocolon were also invaded by neoplasm. The bowel itself was intact. The aortic lymph glands formed a nodular mass which pressed down and almost occluded the inferior vena cava. The abdominal surface of the diaphragm was studded with small white nodules.

The liver was enlarged and weighed 2,900 grams. There was an increase in periportal connective tissue. There were no neoplastic deposits. The hylar glands were free.

The spleen contained three firm whitish nodules which on section proved to be neoplastic.

The pancreas also contained secondary nodules.

The kidneys, ureters, bladder and prostate were normal.

Diagnosis:

Bronchogenic carcinoma with massive secondary deposits producing abdominal symptoms.

Discussion

When first seen in the O.P.D. he was sent up for a gastric series because his complaints were referable to the stomach and he had an indefinite mass in the L.U.Q. For practical purposes X-ray ruled out carcinoma of the stomach.

Five days later he had oedema of the lower extremities, ascites and distended abdominal veins. Although he was breathless his trouble did not seem to be cardiac for he could lie flat without further discomfort. He had no complaints referable to chest. Physical examination was negative, perhaps due to a degree of emphysema which he had.

Such a picture could fit in with a diagnosis of Cirrhossis of the liver. It would account for his gastric complaints, ascites, oedema of the lower extremities, the previous loss of weight and the distended abdominal veins. However, the mass felt in the L.U.Q. seemed to be nodular. The liver was not characteristic. This mass in the L.U.Q. may have been taken for spleen or even kidney. A nodular markedly enlarged spleen is never found in cirrhosis. Furthermore, it is rare to have extensive secondary involvement of spleen in malignant disease unless the primary be in the liver. This man had no jaundice or anything to suggest primary malignancy of the liver. Cirrhosis of the liver and carcinoma of the liver were ruled out.

The possibility of a kidney tumour was ruled out because the urine was always quite clear. Kidney tumours may occur without blood in the urine (15%) but it is unlikely that one of this size would produce no urinary symptoms or signs. Terminal elimination of blood urea is common and is insignificant.

The mass in the L.U.Q. was taken then to be neither spleen nor kidney, but rather a mass of glands, probably due to secondary carcinoma.

Examination of the fluid taken from the abdomen yielded no useful information. The presence of chylous ascites was merely a point

of interest although it indicated obstruction of the thoracic duct—another point in favour of secondary glandular involvement.

It was thought unlikely that a growth in the large bowel could produce such massive secondaries and produce no change or disturbance in bowel habit. Rectal examination was negative—so carcinoma of the bowel was ruled out.

The possibility of a blood dyscrasia was ruled out by careful examination of the blood. He had no suitable superficial lymph glands for biopsy to rule out Hodgkin's disease and lymphosarcoma.

Cirrhosis of the liver, gastric carcinoma, carcinoma of the bowel, kidney turmour and blood dyscrasia having been eliminated, some other explanation for the findings was necessary.

During his stay in hospital this man ran an unexplained fever. This suggested that a primary growth may be in the lung. The diagnosis was fairly clear when the radiologist reported an infiltrating process in the R.U.L. It is known that lung cancer grows insidiously and often the first evidence is extra thoracic metastases. He had none of the cardinal symptoms of carcinoma of the lung. He did have pain referred to the epigastrium and unexplained fever. The problem in this case was to place the primary growth responsible for the massive abdominal secondaries. The line of reasoning followed is presented in this brief discussion. Some of the possibilities were not 100 per cent ruled out but often a 100 per cent diagnosis is not made till the body reaches the post mortem table, and sometimes not then.

What are Your Diagnoses?

1. Leather-bottle stomach
2. Sabre shin
3. Flail joint
4. Student's elbow
5. Nutmeg liver
6. Flea-bitten kidney
7. Thrush-breast heart
8. Saturday-night paralysis
9. Worm-eaten skull
10. Horse-shoe kidney
11. Strawberry gall-bladder
12. Sago spleen
13. Soapsuds (X-ray of bone)
14. Pipe-stem arteries
15. Poker back
16. Currant-jelly stool
17. Hour-glass stomach
18. Collar-stud abscess
19. Hob-nailed liver
20. Chauffeur's fracture

Fill in your diagnosis then turn to page 78 and take 5 marks for each correct answer.

DIFFERENTIAL DIAGNOSIS OF MUSCULAR WASTING

By E. LAMPERT '41

Muscular wasting may occur merely as part of a general wasting of the whole body, due either to chronic lesions such as cancer, tuberculosis, ulcerative colitis, cirrhosis, or to more acute processes such as typhoid fever, dysentry, cholera. In these cases the wasting is generalized, presents no neurological signs and is explained by the underlying disease. Local muscular atrophy may occur due to disease of the parts beneath, such as the shoulder muscles in pulmonary tuberculosis, the muscles about joints in which a pathological process is occurring, splinted joints, etc. No reaction of degeneration (R.D.) occurs in these muscles.

Aside from these general and local causes, muscle wasting may be either due to (1) *disease of muscle*, or (2) *lesion in lower motor neurone.*

Primary Myopathies

(*Progressive Muscular Dystrophy*)

A myopathy is a chronic progressive wasting of the skeletal musculature. The salient features of the primary myopathies comprise heredofamilial incidence, gradual onset in early life, proximal more than distal involvement, occasional pseudohypertrophy, loss of deep reflexes, absence of fibrillation and of qualitative electrical disturbance. There is no evidence of involvement of the nervous system.

Their probable heredodegenerative nature accounts for the frequency with which atypical combined or transitional cases are encountered. Because of this, a review of the literature reveals a maze of nomenclature which, to the student, presents an almost hopeless array of meaningless names. An anatomical-clinical terminology is conducive to a much clearer appreciation of the disease but since common usage demands it, the following classification is set forth.

1. *Primary muscular dystrophy* (usually of lower limbs).
   - (*a*) Pseudohypertrophic form.
   - (*b*) Facio-scapulo-humeral (Landouzy-Dejerine).
   - (*c*) Erb's juvenile type.
   - (*d*) Distal type of Gowers.
   - (*e*) A legion of limited, transitional and late forms.
2. *Myasthenia Gravis.*
3. *Amyotonia congenita* (Oppenheim)
4. *Dystrophy with myotonus.*
   - (*a*) Myotonia dystrophica.
   - (*b*) Myotonia congenita.

1. *Primary Muscular Dystrophy.*

A primary muscular dystrophy is a chronic progressive wasting of the skeletal musculature frequently associated with pseudohypertrophy. Pseudohypertrophy occurs when the increase of fat and fibrous tissue proceeds more rapidly than atrophy of the contractile elements. In any event, whether pseudohypertrophy or atrophy occurs, there is a diminution of the contractile elements of the muscle and therefore weakness is an outstanding feature. The atrophy may, and usually does, affect first the axio-appendicular groups of muscles, namely, the hip and shoulder girdles; it may begin as in the rare infantile cases, in the face and scapulohumeral-region; it may affect the back muscles severely; it may occur in any group of muscles.

The essential character is determined by the fact that the muscles themselves are primarily or objectively the seat of the disorder. Therefore no fibrillations precede or accompany the atrophy; there are no sensory changes and no qualitative electrical disturbances.

The disease usually begins early in childhood, but sometimes as late as puberty or in adolescence. Very often several members of one family are affected.

As the weakness frequently begins in the hip muscles, the first symptom often

is a waddling gait—"He straddles as he stands and waddles as he walks." Soon the glutei and back muscles begin to atrophy and the child has difficulty in sitting up or getting up from a reclining position. The child must eventually roll on its abdomen, try to get up on all fours and climb up on its knees. When the atrophy becomes very severe the patient needs other support in rising, and ultimately he cannot rise at all. Scoliosis, and especially lordosis, which is most marked on standing, are very characteristic.

As the disease progresses the thigh muscles become weak. Soon those of the should girdle and of the upper arm are affected. The arms hang limply, the scapulae cannot be fixed and are winged. Sometimes the shoulder girdle is affected first along with the face (Landouzy-Dejerine). The distal musculature, that is, of the hands and feet, is usually spared, but it begins in these muscles in the rare distal type of Gowers. Rarely the tongue, palate, and muscles of mastication are involved. Even the heart muscle has been shown to be affected. When the facial muscles are involved, there results a special myopathic facies: the wrinkles disappear, the nasolabial folds flatten out, the palpebral fissures are widened, the upper lip protrudes (*bouche de tapir*), the chin is pointed, and the mouth widens transversely during laughter.

The atrophy is usually bilateral, often symmetrical. The deep reflexes gradually grow weaker and are ultimately lost. As the disease progresses quantitative but not qualitative electrical disturbances appear. Rounded, sometimes ball-shaped, prominences lying within an otherwise atrophic muscle belly may be noted. Sometimes these prominences only occur when the muscle contracts, in which event it is a pathognomonic sign of muscular dystrophy.

2. *Myasthenia gravis.*

Myasthenia gravis is only mentioned here to say that wasting is an unusual occurrence and takes place only when there is a permanent paralysis. It is a chronic malady of adult life characterized by a variable paralysis of muscles which is produced or rapidly increased by exertion and which tends to disappear slowly during rest. A permanent paralysis which shows no improvement with rest follows the variable paralysis. This permanent paralysis may be very local in distribution, and may be associated with atrophy of the muscles. The affected muscles on strong faradization soon cease to respond to faradism but remain excitable by galvanism.

3. *Amyotonia congenita* (Oppenheim's disease).

Much controversy wages about the nature of this condition but in this paper it is didactically being placed in the category of the myopathies. It is a rare, probably congenital disease, occasionally familial, that is characterized by extreme flaccidity, weakness without paralysis, slim musculature, no fibrillation, loss of deep reflexes, and a marked diminution of the electrical response. The mentality is normal, speech is unaffected; there are no sensory or sphincteric disturbances. The great relaxation of the muscles and ligaments allows of the most fantastic attitudes being assumed without pain. All the symptoms tend to improve in the majority of cases.

4. *Dystrophy with myotonus.*

Myotonia is a symptom. It consists of a heightened muscular irritability and contractility and a lessened or slowed power of relaxation. Clinically it is characterized by inhibition of voluntary movement through the sudden contraction of a group or several groups of muscles and their failure to relax promptly. Voluntary effort to overcome the sudden accession of tonus is momentarily ineffectual. Only after repeated, slow attempts does execution of movement become possible. Emotion, fatigue, and especially cold, increase or precipitate the myotonia. The myotonia may involve any voluntary muscle. The myotonic reaction consists in a very long, lasting contraction when the muscles are stimulated with every form of stimulus.

4a. *Myotonia dystrophica.*

This is a disease of familial incidence which begins usually in the 3rd or 4th decade, characterized by atrophy of special topographical distribution in which the myotonic symptom is often present. When typical there is atrophy of the face, temporals, masseters and sternomastoids resulting in a "hatchet-face" and round pole-like neck. The muscles of the hands and tongue are frequently affected. Sometimes there is atrophy of the vasti, dorsi-flexors of the feet and the peronei. Any muscle may be affected.

There is no loss of sensation, the tendon reflexes are diminished or lost, fibrillation is absent. Presenile cataract occurs in more than half the cases. The familial incidence of the disease may be shown by a family history of presenile cataract. Other familial stigmata that may be found in the patient as well as the family are premature baldness, atrophy of the testicles, loss of sexual power and general bodily wasting.

The condition progresses very slowly but rarely progresses beyond the age of 45.

4b. *Myotonia congenita* (Thomsen's disease).

This is a congenital familial disease which is characterized by myotonia from early age. The myotonus results in slowness, clumsiness and awkwardness of movement with a great tendency to fall if the balance is upset. Muscular atrophy is rare in this disease, in fact, the rule is a *fine* physique although the muscles are weak.

Lesions of the Lower Motor Neurone

This results in complete flaccid paralysis, absent tendon reflexes, wasting and R.D.

1. Lesions of the anterior horn cells.

A. *With fibrillation.*

(*a*) Progressive muscular atrophy.
(*b*) Amyotrophic lateral sclerosis.
(*c*) Syringomyelia.
(*d*) Peroneal muscular atrophy.

B. *Without fibrillation.*

(*a*) Acute anterior poliomyelitis.
(*b*) Compression of the spinal cord.

1, A (a) and (b).

Progressive muscular atrophy and amyotrophic lateral sclerosis are pathologically similar. In both diseases there is degeneration of the motor tracts, both upper and motor neurones, from the Betz cells to the ends of the axones of the lower motor neurons However, there is no correspondence between the anatomical lesion and the symptomatology, and therefore the distinction between progressive muscular atrophy and amyotrophic lateral sclerosis is purely a clinical one.

Progressive Muscular Atrophy.

Progressive muscular atrophy most commonly begins in the third and fourth decades and is three times as common in males as in females. Sensory symptoms are often present in the beginning and consist of stiffness, dull aching pains, crampy pain, numbness and rarely tingling in the affected muscles. These sensory phenomena usually disappear as the disease progresses and muscular wasting makes its appearance.

Muscular atrophy commences most frequently in the upper limbs when the distal (intrinsive muscles of the hand) or the proximal muscles are first affected in an equal number of cases. The atrophy always commences first on one side but tends ultimately to become symmetrical. The bulbar, neck muscles and lower limb muscles are affected in that order. The wasting is accompanied by loss of power, loss of tone, absent tendon reflexes, R.D. and fibrillation. It is the loss of power and with it the loss of function of the muscles that most frequently bring the patient to the doctor. This is particularly true when the bulbar muscles are affected first.

Amyotrophic Lateral Sclerosis.

Amyotrophic lateral sclerosis is merely a variant of P.M.A. wherein the lesions of the upper motor neuron become manifested clinically. Thus there is atrophy of muscles, with fibrillation com-

bined with hypertonus and hyperactive reflexes. As a rule the upper limbs present more of the picture of lower motor neuron lesion while the lower limbs are predominatingly spastic, ankle clonus and a Babinski response being present in most cases. The abdominal reflexes are most frequently absent. As in P.M.A., there are no objective sensory changes. The bulbar group of muscles, namely, the tongue, lips, palate and pharynx, are frequently affected.

1. A (c). *Syringomyelia.*

The pathological process most frequently affects the lower cervical and upper thoracic segments. It may occur in the medulla, when the disease is known as syringobulbia. The first symptom is a disturbance of sensibility known as the dissociated sensory loss, i.e., sensations travelling in the commissures only are destroyed, while sensations travelling by an uncrossed path are spared. Therefore pain and temperature sensibilities only are lost and these are lost in the segments involved pathologically, most frequently bilaterally in the fingers, hands and chest. The slow destruction of the anterior horn cells gives rise first to fibrillations, then atrophy. Ultimately complete flaccid paralysis develops in the muscles innervated by the affected segments. Trophic and vasomotor disturbances are common.

1. A (d). *Peroneal Muscular Atrophy* (Charcot-Marie-Tooth type).

This is a familial disease, occurring equally in males and females, usually beginning in childhood, progressing slowly for about 20 years, and then coming to arrest. It is characterized by muscular atrophy, distal in distribution, which always begins in the intrinsic muscles of the feet producing pes cavis and retraction of the toes. The atrophy spreads proximally in a horizontal plane and may reach as high as the middle of the thigh or be arrested at any level below that. When the level is below the middle of the leg it gives rise to the inverted "fat bottle calf". The intrinsic muscles of the hand may waste and the wasting may go as high as the middle of the forearm. Slight loss of deep sensibility, loss of vibration sense and tactile loss may frequently be detected. The knee jerk is always present, the ankle jerk frequently lost or diminished. R.D. is present as is fibrillation, while the disease is active.

1. B (a). *Acute Anterior Poliomyelitis.*

This is an acute generalized infection due to a virus characterized by an onset with moderate fever, headache, lethargy, stiff neck, and which may or may not go on to paralysis. If paralysis occurs it is of the flaccid type, the muscles to be affected are painful and sore for about 3 to 4 weeks. The muscles affected are most frequently those serving coarse movement. The paralysis is asymmetrical. Any muscle which shows a response to faradism three weeks after the onset will completely recover and recovery is possible up to two years. The loss of tone, deep reflexes, and power is proportionate to the atrophy.

1. B (b). *Compressiom of the Spinal Cord and Nerve Roots.*

The causes are many and include caries of the spine, wounds of the spine, vertebral tumors, meningeal tumors and cysts, intramedullary tumors, epidural abscess, arthritis deformans, aneurism of the aorta eroding the bodies of the vertebrae, Paget's disease of bone, syphilitic pachymeningitis, and a host of other rare causes. Wasting as it occurs in these conditions, only occurs in those muscles receiving the same segmental supply as the segments of the cord involved and is usually a late symptom. In general, sensory changes and pyramidal tract involvement manifests itself long before wasting. Block of the subarachnoid space may be determined by the Queckenstedt test. Often the Froin or Nonne syndrome is present in compression, and this consists of increased globulin in the C.S.F. absent or slightly increased cell count, and Xanthochromia.

2. Lesions of the Peripheral Nerves

i. *Local Lesions of Nerve Roots and Nerve Trunks.*

(a) Interstitial neuritis.

(b) Cervical rib.
(c) Obstetrical palsies.

ii. *Peripheral neuritis.*

2. Lesions of the peripheral nerves, if complete, manifest themselves in the muscles as lower motor neurone paralysis along with loss of sensation in the part supplied by the affected nerve. To diagnose such a lesion requires an accurate knowledge of the anatomy of the peripheral nervous system. Of course, one may be immensely aided by the history of trauma or its observation in the region of injury. Complete severance of peripheral nerves occurs in gunshot wounds, knife wounds, fractures in which the raw edge of a bone severs a nerve, etc.

2. ii (a). *Interstitial neuritis.*

This is a condition most commonly met with in early adult life and more predomininately in males. It may attack any peripheral nerve trunk and is ushered in with pain in the distribution of the nerve trunk, tenderness of the nerve trunk, pain on stretching the nerve, muscular fibrillation and cramp. Muscular paralysis or loss of sensation is rare and occurs only in the severest of cases, although paresthesia is common. Muscular wasting usually occurs, but it is a general wasting of the painful region, not confined to the distribution of the nerve involved, and therefore resembles the wasting seen in joint disease. Sciatica is the most common form. Brachio, femoral and facial nerve neuritis are other types met with.

2. ii (b). *Cervical Rib.*

It must be remembered that cervical ribs may be present and no symptoms occur, and the picture seen in cervical rib may be present without such an accessory rib being present. This may be due to post-fixing of the brachial plexus or ossification of the tendons of the scalene muscles. Sometimes a fibrous band extends from the transverse process of the 7th cervical vertebra to the first thoracic rib and strangles, as it were, the cords of the plexus.

The symptoms of cervical ribs come on in adult life. Although cervical rib is frequently bilateral, the symptoms are generally limited to one side, usually the right. Pain in the arm is the first and most constant symptom. This may be dull, aching, sharp, or boring. It is usually located on the inner or ulnar side of the forearm and hand, that is, in the radicular distribution of C8 and D1. Dysesthesias, such as numbness, tingling, and a feeling of coldness, often accompany the pain. Objective sensory disturbances, especially impaired touch, pain and temperature become evident as the condition progresses. Soon motor symptoms appear, at first there is weakness, later atrophy and paralysis of the small muscles of the hand, simulating ulnar nerve paralysis. The motor symptoms are generally less marked than the sensory. The arm may feel cold or cyanotic. Frequently the radial pulse is smaller or cannot be felt at all on the affected side, especially if the arm is raised. Horner's syndrome is very rare because the lesion is distal to the sympathetic rami. Occasionally the cervical rib may be palpated.

2, i(c). *Obstetrical Paralysis.*

These may be facial paralysis from forceps; hemiplegia from laceration of the brain substance or haemorrhage; fracture dislocation of the spine with transverse lesions of the cord; injury to the peripheral nerve trunks at the elbow, axilla or groin, in using traction with the finger; and injury to the brachial plexus from the separation of head and shoulder. The facial paralysis invariably recovers within a few weeks. The hemiplegia results only in disuse atrophy if the child survives. Injury to the brachial plexus is the common injury producing atrophy. This is of two main types, the upper arm type of Duchenne-Erb, and the lower arm type of Klumpke, of which the former is the more common.

In Erb's paralysis the arm lies limply by the side; it cannot be abducted or rotated outward, and the forearm cannot be flexed or supinated, i.e., D5 and 6 are affected. Sensory signs are generally slight or absent. Atrophy of the affected muscles is marked but vasomotor disturbances do not occur.

In Klumpke's type of paralysis, C8 and D1 are most affected, leading to atrophy of the small muscles of the hand and the flexors of the forearm.

2. ii. *Peripheral Neuritis.*

In general the first symptoms are those of subjective sensory disturbances such as numbness, tingling, burning, coldness, sharp lancinating or boring pain. Objectively there is a distal sensory loss commonly referred to as stocking and glove anaesthesia. There is tenderness of the muscles on deep pressure. The paralysis has the same distal distribution as the sensory loss. If the paralysis is prolonged more than a few weeks there is wasting, flaccidity, decreased or absent reflexes and R.D. In the early stages the reflexes may be exaggerated. There may be trophic changes in the skin, the nails become curved and brittle, the skin is thin, shiny, may be blue and excessive sweating may occur. In chronic types the subcutaneous nerves may become palpable and contractures, due to fibroiss and overactivity of other muscles, may occur. In certain types of polyneuritis, as that due to lead poisoning, there may be no sensory involvement, only the motor side being affected.

Answers to Questions on Page 72

1. *Diffuse infiltrating carcinoma.* The entire stomach is involved and is very small and thick-walled, stopping abruptly at the pyloric ring.
2. *Congenital syphilis* or *severe rickets.* The tibia presents a marked forward curve which may be also seen in Paget's disease of bone but the subcutaneous border is not as sharp as in the diseases mentioned.
3. *Charcot's disease of joints* which affects the large joints of the lower limb (commonly the knee) in tabes dorsalis and of the upper limb in syringomyelia.
4. *Traumatic bursitis* of the olecranon bursa caused by chronic and repeated irritation.
5. *Chronic venous congestion.* The cut surface shows a mottled appearance of dark brown (congested centre of lobule) and light yellow areas (fatty peripheral part).
6. *Embolic glomerulonephritis* in subacute bacterial endocarditis or *malignant nephro sclerosis.* Scattered over the surface are numerous small red points representing small hemorrhages into the capsular space.
7. *Fatty degeneration* of the myocardium, seen in severe anemias, especially of the pernicious form. The yellow spots represent areas of fatty change.
8. *Temporary paralysis of the muscles supplied by the radial nerve.* The condition by legend is due to the enjoyment of drunken slumber with the arm over the sharp back of a chair, exerting pressure on the posterior cord in the exilla.
9. *Diffuse syphilitic osteitis.* Gummatous formation and necrosis produce the characteristic eroded appearance.
10. *A congenital anomaly* in which the kidneys are fused together, usually by their lower poles. The writers pass down in front of the connecting bridge of tissue.
11. *Cholesterolosis.* The mucosal folds are loaded down by yellow masses of cholesterol ester giving the appearance of the seeds of a strawberry against the red mucosal background.
12. *Amyloid degeneration.* Small pale translucent areas like grains of sago are seen on the cut surface.
13. *Giant-cell tumor.* The X-ray shows a rarefied, multicystic appearance as if the mass was composed of large soap bubbles.
14. *Medical sclerosis of Monckeberg.* The arteries of the muscular type are affected which are hard and tortuous.
15. *Osteoarthrosis of the spine* (*spondylitis deformans*). The rigidity is due to ossification of the spinal ligaments and the normal spinal curvatures are lost.
16. *Intussusception.* With the onset of colic, fecal material is promptly expelled and subsequently only blood and mucus are passed.
17. *Healed gastric ulcer.* When a gastric ulcer astride the lesser curvature (saddle-shaped ulcer) heals, the stomach is divided into two parts.
18. *Suppurative tuberculous cervical adenitis. The* pus burrows through a small opening in the deep fascia into the more commodious space beneath the superficial fascia.
19. *Portal cirrhosis.* The nodules are composed largely of new cells and the depressions are caused by necrosis of liver parenchyma with replacement fibrosis.
20. *Fracture of the shaft of the radius* due to a sudden hyper-extension of the wrist with a backfire, or to a revolution of the crank striking the back of the wrist.

REGIONAL ILEITIS

By C. C. BURKELL, B.Sc., '41

Historical Background and Definition

In 1932 Crohn, Ginzberg, and Oppenheimer of the Mount Sinai Hospital, N.Y., published an article in the J.A.M.A. in which they described, "a disease of the terminal portion of the ileum characterized by a subacute or chronic, non-specific inflammation of the lower 25-30 cms., of the ileum. The inflammatory process, being of an exudative hyperplastic type, eventually resulted in stenosis and obstruction of the bowel and, not infrequently, in internal and external fistulae". Thus was the original description of the disease which Crohn, *et al.*, termed *Terminal Ileitis*. Until that time the description of many granulomatous lesions of the intestines had appeared in the literature, but the group was a heterogenous mixture of both clinical and pathological types. The frequency of a definite clinical pattern, in their collection of 14 cases, induced them to describe the disease as a "clinical and pathological entity". However, since the original description, Crohn, as well as many other observers, has found that the disease may spread to include the caecum, or, may involve the jejunum as well as the ileum. As a result of these new observations, a variety of different names has been suggested, such as, chronic cicatrizing enteritis, regional enteritis, and regional ileitis. The last mentioned term is the one most commonly used and will be used in this paper.

Etiology

Numerous etiological factors have been discussed but the true nature of the condition remains unsettled. There does not seem to be any single etiological factor. In some cases the pathological lesion suggests a tuberculous infection, while in others, it suggests a lymphogranulomatous process. Various types of bacteria have been recovered from the affected tissues, yet, in some cases, it was impossible to culture any organism. No type of bacterium was isolated consistently enough to be called the causative organism. It is interesting to note that, in one case studied by Dr. R. E. Pumphrey of the Mayo Foundation, guinea pig innoculations from the affected segment and regional lymph nodes were positive for the tubercle bacillus. This patient, however, gave a negative Mantoux cutaneous test, and an x-ray plate of his chest was negative for tuberculosis. The disease has been observed in 3 pair of siblings which suggest a common infecting agent. In one incident two brothers were affected simultaneously. It has been suggested (Brinsburg and Oppenheimer) that vascular damage, such as might occur in the presence of repeated self-reducing volvulus or intussusception, might possibly be a causative factor. Clinically, it resembles chronic ulcerative colitis in many respects, except different portions of the intestines are affected.

Clinical Picture

The disease usually affects young adults, the average age being 30 years. It affects males oftener than females in the ratio of 3:2. The Hebrew race is more frequently affected than other races. Out of the 9 cases which have been treated at the T.G.H., since 1934, 6 were Hebrews. Crohn states the disease occurs with almost the same frequency as idiopathic ulcerative colitis, yet only 9 cases have appeared on the public wards of the T.G.H. in the past 6 years.

Four stages may be recognized if the disease is allowed to progress to its full extent. These four stages, with their symptoms and signs, are listed below, but it should be remembered that the underlying pathological process is a continuous one. Hence, at any given time, the clinical picture may be a combination of two or more stages.

1. *Stage of Peritoneal Irritation.*

Symptoms: The symptoms usually develop slowly—over a period of days rather than hours as contrasted with acute appendicitis. There is a colicky pain which is periumbilical at first but later localized in the R.L.Q. The patient may vomit several times.

Signs: The common signs are tenderness, rigidity, and hyperaesthesia localized in the R.L.Q. The patient usually has a slight fever and an elevation of the white blood cell count. Of the 9 cases from the T.G.H., which were reviewed, 5 had a temperature of 100°F. and their white blood cell count varied between 9,000 and 15,000 per cu. mm. It is thus appreciated that regional ileitis closely simulates acute appendicitis. To testify to this fact ten, of Crohn's original 14 patients, had had a previous appendectomy without relief of their symptoms.

2. *Stage of Colitis.*

This stage is often transient or unrecognized. However, it was present in 6 of 30 cases studied at the Mayo Clinic, and 3 of 9 cases at the T.G.H. or an average of 29 per cent in the cases stated.

Symptoms: The symptoms are periumbilical pain and diarrhoea. The diarrhoea consists of three or four loose movements a day. The patient, in this stage, not infrequently, loses weight.

Signs: The signs may be listed as (*a*) tenderness in the R.L.Q.; (*b*) blood either gross or occult in the stool; (*c*) and a low but constant fever.

3. *Stage of Subacute or Chronic Intestinal Obstruction.*

Symptoms: The patient may complain of rather severe abdominal cramps which reach their maximum in the R.L.Q. The cramps, which are associated with borborygmus, are worse following a meal and are relieved by catharsis. The patient may have alternating constipation and diarrhoea. Vomiting is common in this stage. The patient's appetite is poor and his weight loss is progressive.

Signs: The commonest sign is a palpable mass in the R.L.Q. (23 out of 30 cases at Mayo's; 5 out of 9 cases at the T.G.H.). The abdomen is somewhat distended, chiefly below the umbilicus. Peristaltic movements can often be visualized.

The three stages, so far described, often overlap, hence Meyer and Rosi state, "the association of the symptoms and signs of an acute appendicitis with those of an acute but incomplete intestinal obstruction is very suggestive of an acute regional ileitis".

4. *Stage of Adhesions and Fistulae Formation.*

The patient presents himself with a persistent fecal fistula which has occurred spontaneously or has followed an appendectomy. The fistula may manifest itself either as an internal or external variety.

Pathology

The pathological process is continuous and progressive but four stages, corresponding to the clinical stages, may be recognized.

1. *Acute Stage.*

Gross: The serosa is a blotchy red. The mesentery is greatly thickened and contains numerous hyperplastic glands. The terminal portion of the ileum is thickened, soggy, and edematous. The lesion frequently begins just at, or proximal, to the ileo-caecal valve. From there it tends to spread proximally but may extend into the caecum. It is not static as is revealed by the different grades of inflammation which may be found. The cut surface of the intestine is greatly thickened and the mucosa may reveal the beginning of small ulcerations lying in the long axis of the bowel along the mesenteric attachment.

Microscopically: There is some ulceration of the mucous membrane which is covered with a layer of exudate composed of necrotic cells, fibrin, a few erythrocytes, and numerous bacteria. The lymphoid tissue often undergoes marked necrosis. The characteristic cell is an endothelial leucocyte but a few lymphocytes are also present. There is, however, a marked lack of poly-

morphonuclear leucocytes. The submucosa and lymph nodes are edematous. Some inflammatory changes are present in the serosa.

2. *Ulcerative Stage.*

The mucosal ulceration reaches its maximum in this stage. The mucosa becomes necrotic and lost. Under the necrotic layer an atrophic membrane begins to develop. This consists of a single layer of cells with no attempt to restore the secreting glands. Thickening and edema of the bowel still persist.

3. *Hypertrophic Stage.*

Gross: The bowel wall progressively thickens until it is 2 or 3 times its normal size. On examination it is a soggy hose-like mass. The lumen of the bowel becomes encroached upon and distorted. The normal mucosal folds are broken up by the ulcerations and blunted by edema. The ileocaecal valve may be a rigid diaphragm with a small opening. The mesentery is often 2 cms., in thickness.

Microscopically: The mucosa is necrotic or covered with an atrophic membrane. All the layers of the bowel wall, with the exception of the submucosa, show a heavy lymphocytic infiltration. A few plasma cells are also present. Giant cells were common in some cases. These are probably foreign body giant cells attacking particles that were embedded under the regenerated atrophic mucous membrane. During this stage a healing process becomes evident by the fibroblastic reaction.

4. *Fibroserotic and Fistulae Stage.*

The abdominal organs and abdominal wall, which lie adjacent to the inflamed bowel, become involved in the edematous low grade inflammatory process. Subsequently, the healing process forms dense adhesions. The caecum, transverse colon, sigmoid, and anterior abdominal wall are frequently involved. Less frequently the rectum, urinary bladder, vagina, or an adjacent loop of ileum or jejunum may become adherent to the diseased portion. The ulcerative process is progressive throughout the disease and may extend through the adjacent inflammatory and fibrous mass so that a fistula is eventually formed.

Microscopically the picture is one of healing with fibrosis. Many fibroblasts, scar tissue, and scattered islands of lymphocytes is the characteristic picture. An occasional giant cell may be found. This fibrotic process eventually involves the wall of the ileum, the serosa, the mesentery, and regional lymph glands.

Course of the Disease

1. *Spontaneous Recovery.* In acute cases spontaneous recovery may take place. Rosi and Meyer report three, and Erb and Farmer record two cases, which showed gradual improvement after appendectomy. The diagnosis of Regional Ileitis was made at the time of operation. Rosi and Meyer, who followed their cases for 1, 8, and 12 months respectively, state that each case was symptom free and there were no radiological findings at the time of the last examination. Further follow up of cases is essential before it can definitely be stated that complete resolution is possible.

2. *Colitis and/or Obstruction.* The disease may progress beyond the acute stage to the stages of colitis and/or obstruction. The patient is chronically ill. He runs a low grade fever, has a poor appetite, and looses weight. He is incapacitated as far as work is concerned.

3. *Acute Obstruction.* If operation is not performed acute-on-chronic obstruction may suddenly develop.

4. *Fistula and Peritonitis.* Not infrequently, one or several fistulae may form. These have a devitalizing influence and, when external, a dissocializing influence as well. Occasionally a localized abscess may rupture into the peritoneal cavity and lead to peritonitis which may prove fatal.

Diagnosis

1. *In the acute stage* the diagnosis is most difficult for the clinical picture of this disease is not unlike acute appendicitis. However, if associated with the

clinical picture of appendicitis there are symptoms and signs of an acute but incomplete intestinal obstruction, the diagnosis may be suspected.

2. *In the stage of colitis* one must differentiate Regional Ileitis from the other causes of diarrhoea. These causes may be listed as (*a*) the gastrogenic diarrhoea of achylia gastrica, (*b*) the diarrhoea associated with food allergy, (*c*) emotional diarrhoea (all of which may be recognized if a careful history is taken), (*d*) thyrogenic diarrhoea with its associated elevated metabolic rate and concomitant signs, (*e*) carcinoma of the gastro-intestinal tract, (*f*) the dysenteries which are recognized by stool examinations and agglutination tests, (*g*) ulcerative colitis which can be excluded if an examination is made with a sigmoidoscope, or by taking x-ray plates following a barium enema.

3. *Stage of obstruction* with its mass in the R.L.Q. brings to mind appendiceal abscess, intestinal and mesenteric tuberculosis, abdominal Hodgkins, lymphosarcoma, etc., which can only be finally differentiated in the operating room. Carcinoma of the intestine in this region offers a problem but a localized filling defect on x-ray examination may lead to a correct diagnosis. The treatment in both conditions, however, is operative.

4. *When fistulae have developed* actinomycosis must be considered. The failure to find sulphur granules in the discharge makes actinomycosis improbable. Fistulae following an appendicial abscess, tuberculosis, or malignancy must also be considered.

To make the diagnosis of Regional Ileitis one must take a careful history. The slow onset of a disease resembling acute appendicitis yet associated with signs and symptoms of partial intestinal obstruction is suggestive of Regional Ileitis. The patient usually has an appendectomy without complete relief of his or her symptoms, and, after a variable period of time, the patient returns suffering from abdominal cramps and a mild diarrhoea. A mass is palpable in the R.L.Q., peristaltic movements are visualized, and occult blood is found in the stool. It is now that an x-ray examination proves most helpful.

X-Ray Findings

A flat plate taken after a barium meal reveals, as an early sign of Regional Ileitis, distended loops of the terminal ileum. These loops have a fluid level. There is a definite delay in the motility at the end of the small gut. Often contractures of the caecocolon or just the caecum may be seen. The most characteristic finding is the "string sign" of Kantor. This sign does not appear until the lumen of the bowel has been encroached upon by the thickening of its walls. The terminal portion of the ileum will show a marked filling defect, and is represented by a thin string of barium connecting the normal portion of the ileum above to the caecum below.

Treatment

1. In the acute phase the disease is not fully recognized until the abdomen has been opened. Meyer and Rosi believe that one is justified, in certain cases, in leaving the affected ileum alone for, they believe, spontaneous recovery may occur. "If, however, the mesentery is thickened, indurated, and edematous, the ulceration may have extended into the mesentery and hence resection or a short circuiting operation is the better procedure." In any case they remove the appendix to simplify subsequent diagnosis.

2. In the chronic phases of the disease resection is the operation of choice. In performing this operation the surgeon must be certain that he is well beyond the affected area in order to avoid recurrences.

3. When fistula have developed the tract must be dissected out, and the affected segment of the ileum removed.

Conclusion

Although, as already stated, the disease is not common, there have been over 600 cases recorded in the literature since the original description of the disease in 1832. Many of those patients

had suffered chronic ill health for as long as 5 and 10 years. The results of surgical treatment are good, practically all the patients quickly regaining their normal health subsequent to the operation. The early recognition of the disease, together with prompt and suitable treatment, is, therefore, highly advisable.

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"The Manner of Catching Cold, or How Perspiration is Obstructed"

"In sickly, studious, and sedentary People and those of weak Nerves, where the Spring of the Coverlets and Scales, that defend the Mouths of the perspiratory Ducts is weak, the Perspiration little or next to none at all, and the Blood poor and sizey; the nitrous and watery Particles of the Air get a ready and free Entrance, by these Ducts into the Blood, and by breaking the Globules thereof, coagulating and fixing its Fluidity, quite stop the Perspiration, and obstruct all the capillary vessels, the cutaneous Glands, and those of the Lungs and alimentary Passages, when such Bodies are long exposed to such an Air: And thus begets all these Disorders in the Body, that Air thus and thus poison'd, was shewn capable to produce. So long as the Perspiration is strong, brisk and full, 'tis impossible any of these disorders should happen; because the force of the perspiratory steams outward, is greater than the Force whereby noxious Mixtures enter;"

An Essay of Health and Long Life
By George Cheyne, M.D.
Printed for George Strahan, at the Golden Ball over-against the Royal Exchange in Cornhill; and J. Leake, Booksellers at Bath, London, 1725.

THE PROBLEM OF RHEUMATIC CARDITIS

By D. P. BRYCE, '42

It is necessary at the outset to have some conception of the significance of rheumatic heart disease. Current literature most strikingly and constantly compares rheumatic fever with tuberculosis, with regard to incidence, importance and treatment. Certainly a wider public appreciation of this fact is necessary. Rheumatic fever alone accounts for more heart disease in the third and fourth decade than any other one agent, probably about 40 per cent, and in adults this is the greatest single cause of death. Aside from congenital malformations it is the only cause of heart trouble in children. It has been estimated that from 60 to 80 per cent of those with rheumatic fever in childhood ultimately manifest clear signs of heart disease. In many cases an attack of rheumatic fever in childhood forms only one of a long series of progressively damaging events going on through adolescence to adult life, and bringing with it invalidism and early death.

It is not the object of this paper to present rheumatic carditis as a complication of rheumatic fever. Rather is it to be considered as the most important aspect of a general disease. As such is the case, it is necessary therefore, to present a brief sketch of the disease as a whole so that the role of the heart may be viewed in its proper context.

Rheumatic Fever

Rheumatic fever is a disease of obscure etiology, perhaps due to a streptococcus, or perhaps a virus or even an allergic tissue reaction. It is more common in the temperate and northern climates and is more frequent during the winter months.

It is a general infection, often ushered in with sore throats and upper respiratory infection, but sometimes there are no premonitory symptoms. General malaise, fever ranging from 102-104 degrees, rapid pulse, sweating and prostration are common signs and symptoms, along with anemia, mild leucocytosis and skin rashes of various kinds.

Flitting, acute inflammation of the joints, causing extreme pain but usually passing off with complete recovery, forms a typical picture of one secondary aspect of this disease. Chorea, indicative of cerebral involvement, and distinguished by uncontrolled movements of skeletal muscle with ataxia and weakness, is also common. Such findings may be confined to one side of the body or may be bilateral. It is interesting to note that the case marked by chorea often shows heart lesions which are much milder than usual. Particularly in the British Isles, subcutaneous nodules representing collections of the familiar Aschoff bodies form a common sign. Finally and very rarely a mild nephritis with urinary signs, pneumonia and pleurisy are indicative of the widespread nature of this disease.

Rheumatic fever is a disease of youth, and the age factor has an important bearing on prognosis. In childhood the heart suffers; beyond twenty years of age an initial attack confines itself to joint lesions for the most part. In its great tendency to recur lies the danger to the heart. Recurrent childhood disease will almost inevitably result in permanent myocardial damage. Another feature of importance is the quiet subclinical course of the disease in many cases. Such patients develop cardiac lesions insidiously.

Thus rheumatic carditis forms the main topic of this paper. For purposes of classification it will be divided into early or acute cases, and the late or chronic types.

Early Cardiac Involvement

There are two periods in childhood when cardiac damage following an attack of rheumatic fever is most common. These are from five to seven and from nine to ten years of age. The onset of an attach varies from the acute febrile

polyarthritic type, to a type with more general symptoms, fatigue, pallor, occasional sore throat, aching joints and irritability. Such an "unwell" child should be considered rheumatic until proven to be otherwise.

Signs and Symptoms

1. The first sound at the apex is commonly short and soft, and it is particularly suggestive if softer than the second sound at this area.

2. A mitral systolic or an aortic diastolic murmur or both may be present, and if so, are indicative of myocardial infection or degeneration with dilatation of the heart. In this respect it must be kept in mind that there is a functional systolic murmur heard at the apex, in 70 per cent of normal children.

3. Also at the apex a third heart sound may be detected, which will cause the typical "gallop" rhythm in this area. A mid-diastolic or presystolic mitral murmur may be heard. In considering mitral stenosis as a possible factor in the production of this murmur it must be remembered, that mitral stenosis requires at least five years following infection for development.

4. The electrocardiogram is useful in detecting delay in conduction through an affected heart muscle. Such points may be prolongation of the PR interval or changes in the QRS and T waves indicating a transient intraventricular conduction defect.

5. A leucocyte count ranging from ten to sixteen thousand.

6. Signs and symptoms of pericarditis. Here the patient may suffer from dyspnoea and gastro-intestinal symptoms, and show signs of enlarged cardiac dullness with a characteristic to and fro friction rub. There may be precordial pain and tenderness and rarely an apparent area of pulmonic consolidation at the tip of the left scapula.

7. The pulse rate forms a very valuable guide, and as such is more important than the temperature. The pulse rate is always elevated in an active disease with the occasional exception of a temporary fall to normal.

Pathological Findings

These will be discussed under general pathological processes affecting the heart as a whole. It may be emphasized here that of all the cardiac lesions, those changes occurring in the myocardium are by far the most important, and may be the cause of death in the absence of all other signs or lesions.

A. Non specific changes: These are similar to those appearing during other infective toxemias. They are edema and leucocytic infiltration, with fibrinlike change in the connective tissue, and are seen in the pericardium, muscle, and affected valves. They are collectively known as the "Arthus phenomena."

B. Myocardium: Granular, fatty and hyalin degeneration is common in the myocardial fibres. Atrophy and more rarely necrosis is also seen.

C. Specific Rheumatic Granuloma: These are of course the familar "Aschoff" bodies which are found in the periarteriolar connective tissue framework. Even in rapidly fatal cases typical, fully formed, nonfibrosed Aschoff bodies are found. Such reaction is seen in a less compact form in the valvular lesions.

D. Pericardium: Here there are the typical inflammatory changes in a serous membrane, with exudation of large quantities of fluid in the more severe cases. Heavy deposits of fibrin are common.

E. Vascular Lesions: These are usually non-specific lesions of the coronary arteries. The changes are oedema and hyperphasia of the intima and adventitia with occasional nodules in the latter, and oedema and hyalin degeneration in the media. These lesions vary from case to case and do not correspond with the severity of myocardial damage. They are probably quite rare, and in any case as the attack is usually not fatal, they clear up completely.

Significance of cardiac findings during the first attack

In the first place, many cases with such findings will prove to be some disease other than rheumatic heart disease. Also, however, even in cases proven to be rheumatic in origin, many

will be found to show no permanent demonstrable heart disease. However, we do know that recurrence of attacks, particularly many of a mild nature, will almost inevitably result in cardiac damage, and also that the younger the child the more likelihood there is of heart disease. To illustrate, we will present the figures of a recent series. In this survey children with demonstrable clinical heart lesions during their first attack were followed and observed over two decades. Of these children 31 per cent recovered without apparent heart damage. Within ten years 25 per cent of above showed permanent valvular damage, and of these—

Two-thirds had a recurrence of fever or chorea in a typical attack.

In one-third signs appeared insidiously. That is, they suffered from continual subclinical activity of the disease.

During the second ten years only about one per cent, or five patients, showed signs of damage and of these two had a recurrent attack. Thus is demonstrated the significance of recurrences and of active undemonstrable disease. The age factor is also very definitely shown to be of great importance.

From the above picture it is easily seen how the early acute rheumatic heart disease blends into chronic rheumatic carditis.

Late or Chronic Rheumatic Carditis

Here there are two main pictures of onset. There is the patient who has numerous recurrent attacks of fever and arthritis with added cardiac damage at each attack, until death results from heart failure in the course of a severe attack.

The other picture is that of a more insidious and imperceptible increase in cardiac damage with slow but progressive signs of failure and congestion. There may be little or no history obtainable to indicate rheumatic fever.

Pathology

The myocardium shows a slow fibrosis and scarring with early hypertrophy ollowed by dilatation of the heart.

The valves, always the mitral, sometimes the aortic and mitral, show sclerosis and thickening of the cusps and chordae with the typical pin head vegetations and greater or less degree of calcification. Such conditions lead to stenosis or incompetence or both. In the case of mitral stenosis secondary changes are seen, the chief of which is the dilatation of the left auricle.

The pericardium may be thickened and adherent with obliteration of the pericardial cavity.

Patients suffering from chronic rheumatic carditis will come to the physician presenting many diverse pictures and indications of their disease. These can be generally grouped and classified in such a way as to indicate not only signs and symptoms but also prognosis. They will demonstrate the diverse forms of valve lesions, the varying degrees of cardiac enlargement and disordered rhythms, and the different degrees of failure, presented by rheumatic heart disease.

A. The Chief Group

These patients show the typical common group of symptoms and signs associated with late heart disease, which has resulted in extensive cardiac damage. They complain of breathlessness and poor exercise tolerance, and exhibit a definitely enlarged heart, signs of mitral stenosis, and aortic disease. These patients often develop auricular fibrillation and may show some signs of venous congestion. Their prognosis is not good and they usually develop rather sudden congestion with failure. Patients with fibrillation occasionally show embolic and thrombotic accidents arising from thrombus formation in the left auricle.

B. Those cases in which valvular disease is the chief finding

These cases show little or no cardiac enlargement and have a fair exercise tolerance. They will show signs most often of mitral stenosis, and also aortic regurgitation or stenosis. The characteristic murmur, and one which is in itself diagnostic, is the low pitched rumbling mid-diastolic or presystolic murmur heard best at the apex of the heart.

Often an early murmur may only be heard by listening to the first dozen beats, when the patient lying down has been rolled over on his left side. With this lesion to a varying degree are findings such as dilatation of the left auricle and passive venous congestion. These patients have a relatively good prognosis.

C. Auricular Fibrillation the Chief Finding

Auricular fibrillation is an important factor in this disease. Two-thirds of all fibrillators are rheumatic and one-fifth of rheumatic cardiacs fibrillate. It is most common in mitral stenosis and increases in the older age groups until at least half of the cases of mitral stenosis reaching forty years of age are affected by it. The onset of fibrillation is usually quite serious as it throws a heavy weight on the already reduced cardiac reserves, and if these are poor, it may form a terminal picture with congestion. Patients in this class with the aid of digitalis are relatively well off, although their outlook is not as good as class B.

D. Cardiac Enlargement the Chief Finding

These are usually cases of marked aortic regurgitation and have a poor exercise tolerance. They rarely demonstrate severe attacks of angina. Their prognosis is a few years at the best.

E. Venous Congestion the Chief Feature

Congestion usually is the striking feature and one which controls the outlook and treatment. It is most often found with mitral stenosis and varying degrees of enlargement. These cases are of course immediately threatening.

The Chief Diagnostic Problem

From the above it is obvious that diagnosis of a full blown case is not difficult nor is it very helpful. The main hope is to diagnose and separate those early cases in childhood, so that they may be supervised and protected from recurring infection.

Our problem then is early diagnosis. However, it has been seen that with development of typical signs and symptoms the disease will be well advanced. Thus it is necessary to diagnose cases before the usual signs are present. This problem is chiefly one for the X-ray and the electrocardiogram.

Electrocardiogram

1. The finding of a broad flattened or notched P wave especially with right axis deviation.
2. Continued prolongation of PR interval.

These findings are supporting evidence of myocardial damage.

X-ray

The object here is two-fold:

(a) to demonstrate an enlarged left auricle,
(b) to demonstrate calcification of the mitral valve cusps.

The X-ray plate may show in right anterior oblique view, a posteriorly displaced oesophagus due to enlarged left auricle, and in anterior-posterior view show a filling out of the superior concave curve of the left border of the heart, again due to enlarged left auricle. A convex knob representing a dilated pulmonary artery may also be demonstrated.

More important than the X-ray plate is the use of the fluoroscope. It is valuable here in that the observer may separate chambers by their pulsation, and can note any abnormal pulsations. Also he is able to adjust the patient and machine to obtain the most advantageous view. It must be realized also that fluoroscopy is the only reliable method of locating and identifying intracardiac calcification during life, a finding which is almost pathognomonic of rheumatic mitral disease. Unfortunately, this finding is possible only in about ten per cent of cases in life.

Demonstration of enlarged left auricle by fluoroscopy by:

1. A filling out of the upper concavity of the left border of the heart or a double convexity in that region due to enlarged auricle and dilated pulmonary artery.
2. Dilatation of the left auricle pos-

teriorly seen in the middle third of the cardiac shadow in lateral view, or seen by displacement of a barium filled oesophagus.

3. Widening of base of heart due to enlarged left auricle and pulmonary artery seen in antero-posterior view.

4. Dilatation of pulmonary vessels in the lung roots due to stasis of pulmonary circulation.

5. Hypertrophy of the right ventricle seen in the left anterior oblique view which demonstrates a convexity of the right border posteriorly.

6. Narrowing of the precardiac space in the lateral view.

7. In advanced cases the left auricle may displace and obstruct the left bronchus and may even form, or project from the right border of the heart.

Rarely this picture may be simulated by patent ductus arteriosus or partial heart failure with dilatation and fibrillation. Nevertheless dilatation of the left auricle is still one of the most definite signs of mitral stenosis, particularly in absence of heart failure.

Demonstration of Calcified Areas in the Mitral Valve

This phenomenon is seen only rarely during life by the fluoroscope.

It is essential first to completely acclimatize the eyes to the darkness. Then look for dancing shadows in the region of the mitral valve as the valves have a to and fro movement of about 2 to 3 centimetres with systole and diastole. The valves are situated just under the line of the auriculoventricular junction which forms an angle of about 45 degrees with the horizontal and is seen best at the left border.

It is wise to rotate the patient to the left to avoid the spine and to have him take a deep breath to avoid pulmonary distortion due to respiratory movement.

There is a differential diagnosis to be made between the aortic and mitral valves and a calcified mitral annulus fibrosus. The following points would be of assistance:

1. The general shape of the heart.

2. In the left anterior oblique view the mitral valve is seen in the posterior third of the heart shadow while the aortic valve is seen in the middle third.

3. The calcified annulus fibrosus is usually U, J or O shaped, and is more homogeneous than the irregular valve plaque.

It has been conclusively shown by following the cases that patients with no demonstrable signs of mitral disease may, if they present a combination of dilated left auricle with calcification of the mitral valve discovered fluoroscopically, be diagnosed mitral stenosis. If the dilated auricle alone can be demonstrated the diagnosis is strongly suggestive.

Thus it is concluded that by routine fluoroscopy in the hands of a trained observer, earlier diagnosis of rheumatic mitral disease may be expected.

The author wishes to express his sincere appreciation for the constructive criticism of Dr. J. Hepburn.

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AMPUTATIONS

By A. W. MAHOOD, '41

The term amputation in surgical literature means the removal of the whole or part of an extremity, including disarticulations or amputations through joints.

In this discussion it is not proposed to go into any precise textbook description of amputations, but rather to discuss things to be done, and things to be avoided when doing them. Particular points of interest in the most useful amputations of the upper and lower limbs will be considered.

To begin with, things of importance which are common to the great majority of amputations will be discussed. First, and foremost in importance is the saving of life, after this the main considerations are those of establishment of function and the gaining of good form or cosmetic effect. After choosing the level for amputation from considering these factors, it is necessary to have a clear idea as to how the various tissues and structures encountered may be handled.

Skin flaps: obviously these must extend far enough below the level of bone section to effect a good closure. It is not considered good surgery to sacrifice needed bone length or blood supply to get ideal skin flaps. These will be dealt with later under individual amputations.

Fascia: should be left attached to skin to preserve blood supply, prevent skin to muscle adhesions, and give good covering for cut end of bone.

Muscle: should be cut longer than bone to allow for retraction.

Nerves: should be cut short and be injected with 95 per cent alcohol above the point of section.

Blood vessels: all bleeding vessels should be ligated. In most cases a tourniquet is used. It is contraindicated in advanced arterial disease.

Bone: a cuff of periosteum ½ inch above the saw line is usually removed, but in the presence of infection this is not done. Bone wax to the end of the bone will usually control haemorrhage.

Sutures: non-absorbable sutures should not be buried in the stump.

In the presence of infection, disarticulation is preferable to bone section.

Most Useful Amputations of Upper Limb

1. *Hand:* Two general principles to be observed:

(a) To obtain maximum function rather than form. Thus the axiom "save all you can" does not always hold true. It is better to have a useful hand than one that looks well but can do nothing.

(b) Amputation should be done through healthy tissue to ensure good early healing and movement.

2. *Thumb:* The thumb is the most useful part of the hand since it opposes all the other digits. A thumb even alone may be of some use in aiding the opposite hand in certain jobs. For maximum usefulness it should be as long as possible.

3. *Fingers:* According to Dr. J. H. Couch the greatest cause for failure in amputations through the fingers is that the skin flaps are not made sufficiently long, so that when retraction occurs the scar breaks down. To ensure a good result both anterior and posterior flaps should be equal in length to the diameter of the finger.

Amputations through the distal phalanges usually give good results. The best cosmetic effect is procured by using a skin graft on the end which saves bone length by requiring shorter skin flaps.

If the index finger has to be amputated above the distal phalanx, the best all round result will be obtained by removing the whole digit, and including the head and part of the shaft of the metacarpal.

An amputation at any level of the phalanges in the middle and ring fingers will usually give a good result. However, disarticulation at the metacarpal-phalangeal joint does not give such a good result, since it removes the support for

the adjacent digits. The metacarpal is forced backwards by the neighbouring metacarpals, and it becomes quite unsteady. As a result, it is better to remove the head and part of the shaft of the metacarpal, rather than to do a disarticulation.

There is practically no disability resulting from amputation of the little finger, so that it may be done at any level. Metacarpo-phalangeal disarticulation here is not good since the hump left is a nuisance. Therefore, as before, it is better to remove the head and part of the shaft of the metacarpal.

4. *Wrist:* Disarticulation here is not good since it cannot be fitted with a satisfactory appliance, and healing usually leaves a cold blue stump which may be tender. Thus amputation higher up is indicated.

5. *Forearm:* In this region there are three outstanding points of interest.

(a) Optimum functional level — As pointed out above wrist disarticulation is not satisfactory, and the same applies to amputations in the distal third of the forearm. Among bad results obtained may be: poorly covered bone ends and deficient blood supply with a cold blue sensitive stump which may ulcerate eventually. The ideal level for bone section is between the middle and lower thirds.

(b) An amputation should not be made less than two inches below the insertion of the biceps since the resulting stump cannot be fitted with an appliance. However, *this alone* should not be considered in doing a forearm amputation since patients use appliances so infrequently in these amputations.

(c) Anterior and posterior skin flaps are used. These are of equal length, each being more than one-half the diameter of the arm.

6. *Arm:* Points of interest.

(a) Avoid disarticulation at the elbow since the stump will not be well covered and will be sensitive. Apart from this try to preserve as great a length of humerus as possible.

(b) In the lower two-thirds of the arm, each skin flap should be three-quarters the diameter of the arm. In the upper third, using a long lateral and a short medial flap, the deltoid muscle may be used to cover the end of the bone.

(c) No tourniquet is used above the middle because of the danger of infection. Instead cut down on the large vessels and ligate them.

(d) Optimum level for amputation is one inch above the epicondyles; and the best method is a tendinaplastic procedure.

(e) If done above this level and through the fleshy part of the arm don't use muscle flaps.

(f) Amputation of the upper third is better than shoulder disarticulation, because it saves the normal shoulder contour, is easier to do, and results in less trouble with the nerves later on.

7. *Shoulder:* Points of interest in disarticulation here.

(a) Reasons for amputation — traumatic injuries, gunshot wounds, neoplasms, and a completely paralysed arm which is a great nuisance to the patient.

(b) Interscapulo—thoracic amputation removes the scapula and most of the clavicle with the arm. Malignant disease is its only indication and even in this it is of doubtful value. Its great risk is loss of blood.

Most Useful Amputations of Lower Limb

1. *Functional considerations; great toe and other toes.*

The loss of one or more toes usually does not cause great disability. Occasionally there is some defect in walking when the great toe is lost. Any other toe apart from the second may be taken off with little or no ill effect. When the second toe is removed there is a tendency towards a hallux vagus deformity later on. Partial amputations of toes are not done apart from the great toe.

Plantar flaps are usually best because of their special structure which permits heavy pressure and so gives a tough insensitive stump.

Interphalangeal amputations are preferred in the great toe. Local nerve block using 1 per cent novocaine and an elastic tourniquet are advantageous.

The head of the first metatarsal deserves special consideration. It is one of the three primary bearing points of the foot, and its loss results in great functional disability; thus, if at all possible, it should be saved. The great difficulty often is to obtain sufficient tissue to cover it.

Amputations of the other toes are preferably done at the metatarso-phalangeal joints.

Another thing to remember is that the tendons of the Flexor and Extensor Hallucis Longus must be fixed somehow, since they are required as important supports for the medial plantar arch.

Rather than leave only one or two toes alone on a foot where they tend to be of more trouble than use, it would be better to remove them all.

Tarso-metatarsal and tarsal amputations and disarticulations are not very important, and are rarely done any more.

2. *Ankle—Syme's amputation.*

(a) Advantages—

i. Provides an end-bearing stump which will support the whole body weight.
ii. Only the foot is removed and thus maximum leverage power is retained.
iii. It is less mutilating than many leg amputations.
iv. It shortens the leg enough to allow the fitting of an appliance with an artificial ankle joint.

(b) Disadvantages—

i. Leaves a thick unsightly ankle.
ii. Appliances are usually heavy and rather clumsy.
iii. Sometimes there is danger of a cold sensitive stump.

(c) Requirements for success—

i. Avoid damage to Posterior Tibial artery and its medial calcaneal branch, also the first parts of the Medial and Lateral Plantar arteries.
ii. The skin flap should be undamaged.
iii. Tibia and fibula must be cut absolutely horizontally to avoid any undue pressure.
iv. There must be no acute infection or discharging sinus in the neighbourhood.
v. The flap should be trimmed to give a snug fit.

3. *Leg:* Requirements for success—

(a) Optimum level — middle third. Above this level save all the length possible; if less than two inches it is probably better to do a Gritti-Stokes amputation to which an appliance may be fitted.

(b) In amputations through upper third the whole fibula should be removed. This prevents a painful pressure point and allows better weight distribution.

(c) Flaps—mid leg—use long anterior and short posterior flaps. Above mid leg use equal antero-posterior flaps to save length. Especially at the optimum level tendinous flaps over the bone ends are good. Above this level the muscles may be cut short and fascial flaps used.

4. *Thigh:*

(a) Gritti-Stokes amputation—advantages over disarticulation at knee. Easier to fit with an appliance, and is short enough that an artificial knee can be put on and both knees will appear to be at the same level, and the limb can be swung forward clear of the ground on walking.

This amputation requires:

i. Absolute aseptic technique for successful implantation of patella on to the end of the femur.
ii. A long anterior flap with the patella is required.
iii. Entire cartilagenous surface of patella must be removed.
iv. Suprapatellar bursa must be dissected out to prevent effusion. Avoid this amputation when there is any infection near at hand, and if any of the skin required for flaps is damaged.

(b) Kirk has described a tendinoplastic operation to be done when a Gritti-Stokes is not feasible, i.e.—when a long anterior flap cannot be obtained, and where amputation is required higher up. This operation gives good results, utilizing the tendon of the quadriceps.

(c) Above lower 1/3 of thigh, ampu-

tations require antero-lateral and postero-medial flaps because—(i) Retraction of hamstrings is greater than that of quadriceps. (ii) The femur lies nearer the anterior and lateral surfaces of the thigh than to the posterior and medial surfaces.

5. Hip—Disarticulation:

(a) Advantages over short thigh amputations:

i. Good functional results may be obtained due to the absence of the chafing seen in short thigh stumps.

ii. A pelvic socket is provided in which the patient sits and is able to work, and walk relatively long distances.

(b) Requirements for success—

i. Avoid having the scar over the Ischial tuberosity.

ii. Prevention of bad effects from blood loss by giving a preliminary transfusion and by using an Esmarch bandage on the limb. This bandage forces the blood from the limb to the body, and results in saving to the body an amount of blood equal to two or three average transfusions. The large blood vessels of the region must be ligated and the surgeon must be prepared to deal with the numerous anomalies and variations which occur in them. A thorough knowledge of the anatomy of this region is absolutely essential to carry out this operation successfully.

iii. Fill the acetabular cavity with a pedicle muscle flap.

(c) A transcervical amputation promotes quicker healing; and other things being equal it may be the method of choice.

The writer is deeply indebted to Dr. J. H. Couch for his kindly help and encouragement.

"On the Discriminate Use of Drugs"

"To carry out the principle of rejecting remedies because they are capable, if used without due discrimination and discretion, of doing harm, would to be emasculate the materia medica. A remedial agent can have but little value if it be not capable of acting injuriously as well as usefully. In the ability to employ potential agencies so judiciously as to secure their good and avoid their evil effects, lies the secret of true success in the practise of medicine."

Medicine of the Future

An address prepared for the Annual Meeting of the British Medical Association in 1886. By Austin Flint (Sr.), M.D.

D. Appleton and Company,
Bond Street, New York.

EPISTAXIS

By J. G. MICKLER, '42

In addition to being incorporated in the war-cry of a prominent and much-respected faculty on our campus, the word "EPISTAXIS" is used also to designate the condition of rhinorrhagia, or more simple, bleeding from the nasal passages. Bleeding from this area is of common occurrence, and may assume alarming proportions, so that a brief discussion of its etiology and treatment should be of use to those engaged in the study of Medicine.

Etiology

Epistaxis is largely caused by erosion or rupture of small bloodvessels in the nasal mucosa, and may be due to local or general causes, or to combinations of these, or may occur spontaneously, with no apparent underlying cause. The nasal mucosa is very richly supplied with blood, and a small lesion is apt to produce a surprising amount of hemorrhage.

A. Local Causes.

a. Trauma: Trauma to the nose itself, the nasal mucosa, or the base of the skull is possibly the most common cause of epistaxis, evidenced by the bleeding following blows, fracture of the skull base, violent sneezing or nose-blowing, foreign bodies in the nose, nasal operations, etc.

b. Ulceration: of traumatic, syphilitic, malignant, tuberculous or leprous character.

c. Neoplasia: such as adenoid growths, polypi of the mucosa, fibroma, angioma or rarely, malignancy.

d. Varicosity: of vessels of nasal mucosa; or multiple hereditary telangiectases.

e. Acute Infective Inflammation: as in severe catarrh, diphtheria, scarlet fever and influenza.

B. General Causes.

a. High Arterial Blood Pressure: as occurs in chronic renal disease, arteriosclerosis with hypertension, gout and cirrhosis of the liver.

b. High Venous Blood Pressure: as seen in acute and chronic bronchitis, emphysema, failure of the right ventricle and also in cerebral congestion, when an emissary vein passes from the superior longitudinal sinus through the foramen caecum to communicate with the veins of the nasal mucosa. Venous blood pressure increase also sometimes produces epistaxis in young people, after very strenuous exercise.

c. Altered Conditions of the Blood: as for instance in hemophilia, purpura, pernicious anemia, scurvy, leukemia, chlorosis and obstructive jaundice, (avitaminosis K) ; and also at the onset of acute specific fevers such as typhoid fever, scarlet fever, measles and rheumatic fever.

d. Decreased Atmospheric Pressure: tends to produce epistaxis, as aviators or mountaineers can relate. Caisson disease, or sudden decompression from high to normal external pressure from any cause, falls into this class.

C. Ideopathic.

Epistaxis without apparent cause is often attributed to "congestion" and occurs: in childhood; at puberty in females as an evidence of vicarious menstruation; and at the menopause.

It is evident that for the sake of completeness and brevity, no attempt has been made, in the above, to set down the physical factors involved in the production of the hemorrhage, but in most of the causes listed the use of a little imagination will reveal these.

Diagnosis

In the investigation of every case of epistaxis, the patient should be carefully questioned regarding previous attacks, or the occurrence of trauma, etc., and the nasal passages must be meticulously examined. It may be found that the hemorrhage is proceeding from a "bleeding point" or is simply oozing from patches of mucosa. The site of election of the bleeding point is variously located as: Little's area, in the region of the antero-inferior portion of the cartilaginous nasal septum, or as an ulcerated spot upon the septum near the junction of ethmoid and vomer. These are the most common sites, but of course, blood may issue from injur-

ies, ulcers, telangiectases, etc., anywhere on the septum, turbinates, roof or floor of the nasal passages. In every patient, too, unless the cause of the bleeding is obviously simply traumatic, the urine should be examined for evidences of renal disease, and the blood pressure determined. Blood usually issues from only one nostril, but may come from both, and in the former case is likely to be due to local causes. It is important to remember that if the patient is lying down, blood passes into his throat and may be confused with hemoptysis or hematemesis. Recurrent epistaxis is also likely to be the result of local causes, as for instance, a small poorly healed area of ulceration easily re-opened by mild trauma, malignant disease, adenoid vegetations, hereditary telangiectases.

The age of the patient is of some use in uncovering the underlying cause of the condition. In infancy, local injury, nose picking or syphilis of the nasal bones commonly produce bleeding. In childhood, falls and blows, foreign bodies, adenoids, heart or blood disease or local congestion are the common precipitating factors. In adult life, almost any general or local cause may be at fault, while hypertension is the usual malefactor in elderly persons.

In any event the diagnosis rests upon a general examination of the state of bodily health and upon a detailed local examination of the nose.

Treatment

In many cases, epistaxis stops spontaneously, but if this does not occur, there are several means at our disposal to stop it.

The simplest method is to have the patient pinch the nostrils together for as long as half an hour, breathing the while through his mouth, and sitting in the upright position, as this reduces the blood pressure at the site of hemorrhage. The patient should be exhorted not to blow the nose for some time after the hemorrhage has ceased, even if clotted blood hampers the breathing. If the hemorrhage is too severe to be effectively combated by these means, the anterior nares may be carefully and tightly packed with ribbon gauze soaked in 1:1000 adrenaline in physiological saline solution. This packing must be removed after 24 hours as it may become infected and produce middle ear inflammation. If the bleeding point can be located, it may be obliterated by the use of fused silver nitrate sticks or by the electro-cautery in the hands of an expert. If the bleeding point be far back towards the nasopharynx, packing of the posterior nares may be effective in compressing it, and is carried out by passing a soft rubber catheter along the floor of the nostril down into the pharynx, where it can be grasped and brought out through the mouth. A strong silk ligature is then tied to the oral end of this catheter and to the other end of this ligature is affixed a pledget of gauze, with an additional 12″ of ligature for purposes of removal. The nasal end of the catheter is then withdrawn and secured, thus forcing the gauze plug into the posterior nasal aperture and compressing the bleeding point.

Other methods that may be employed if the above mentioned materials are lacking are: plugging of the anterior nares with gauze or cotton wrung out in hydrogen peroxide; painting with tannic acid, or silver nitrate solution; and insertion of a finger cot into which has been tied a catheter, by which it can be inflated while in position in the nostril.

Some measure of success has been claimed from the use of 1:10,000 Russell's viper venom solution, mocassin-snake venom, or salt pork application to the bleeding point.

Angiomata and polypi should be removed, the former by electrolysis, and the latter by removing the polyp and part of the underlying mucosa, under local anaesthetic.

In most cases, one of these methods, combined with the co-operation of the patient, secured on the assurance that his blood loss can be controlled and stopped, will be sufficient to put an end to the most profuse and alarming sort of epistaxis.

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INTRAVENOUS THERAPY

By ALLAN J. LONGMORE, '41

In this article we will attempt to deal with the administration of therapeutic materials by continuous intravenous flow. We will not consider transfusion of blood, other than to mention it, nor the intravenous administration of the almost innumerable drugs used for the treatment of specific disease, where these drugs are given in small quantities, by repeated injections.

Intravenous therapy had its scientific origin in the 17th century. Long prior to this, however, it was mentioned in the books of the priests of Appollo, and Ovid in his *Metamorphoses* tells of Medea renewing the youth of the senile Pelias with a "mixture of blood drawn from the veins of a black ewe infused with semen, with the flesh and pinions of a stryx, or the entrails of a wolf."[1] At this time the deed was regarded as a miracle performed by a sorceress. In the 15th century, in Vilari's *Life of Savonarola*, we read of an attempt to prolong the life of Pope Innocent VIII, which not only failed, but also cost the lives of the three young boys who were the donors. There is doubt as to the authenticity of this, since it was not until 1628 that the true circulation of the blood was discovered by William Harvey. In the same year, Giovanni Colle of Padua in Italy mentions blood transfusion, and the idea of including drugs in the blood administered. In 1651 Robert des Gavets in France actually performed the operation. In 1652 Francesco Folli writes of a "tube made of silver for insertion into the donor's artery, and a cannula of bone into the recipient's vein, the two to be joined by a hollow tube formed from the blood-vessel of some animal."[1]

Fifteen years later, Richard Lower gave an account of his own technique in dogs, using the donor's vertebral artery and the recipient's jugular vein. That same year Meyer used two humans, the connection being from vein to vein. In 1700, the median basilic vein was used for injection of medicine. Harwood of Cambridge in 1792 reanimated an exsanguinated dog by blood transfusion; and in 1818 Blundell found it was unsafe to mix the bloods of two different species. Twenty years later Blake wrote an article "on the physiological effects of various agents when introduced into the vein."[1] In 1875 Landois discovered that animal serum would hemolyze human blood. In 1901 the most important work was done, Landsteiner finding that the sera of donors hemolyze alien blood, and Carrel's work on blood-vessels being responsible for our modern technique of blood transfusion and other intravenous therapy. Then came a lag, until Ehrlich's discoveries revived interest in intravenous medication. Finally, in 1913 Lindemann perfected a technique for the indirect transfusion of whole blood and recognized the need of tests for hemolysis and agglutination.

Indications for intravenous administration of fluids:

1. Hemorrhage
2. Dehydration
3. Shock
4. Infection—patient unable take anything per os
5. Toxemias as result of acute infection in children
6. Obstetrical (a) pernicious vomiting
   (b) hemorrhage
   —abortion
   —accidental
   —placenta praevia
   —post partum
7. Alkalosis—due to loss of fluid and acid

Contra-indications:

1. Oedema
2. Cardiac failure
3. Pre-eclampsia, eclampsia
4. Recent coronary thrombosis with cardiac infarction

Fluids used:

1. Whole blood
2. Concentrated serum

3. Normal saline
4. Saline—3 per cent, 5 per cent, 6 per cent
6. Glucose in saline (5 per cent-10 per cent) solution
7. 2 parts 5 per cent glucose to 1 part normal saline
8. Gum acacia
9. Physiological saline

Practically the only place where normal saline cannot be used (in addition to the general contra-indications) is renal failure. In such cases 5 per cent glucose in distilled water is used. Where there is anuria, oliguria, or renal insufficiency, up to 1800 cc. for a woman, and 2000 cc. for a man, of normal saline may be given in 24 hours; if more fluids are needed, use 5 per cent glucose in distilled water. In alkalosis resulting from vomiting, with loss of hydrochloric acid, a large volume of normal saline can be used. If the chlorides in the blood are extremely low, a total of 15-20 gm. of sodium chloride given as 3-6 per cent solution may be used, it being most important to keep a close check on the blood chlorides. Similarly, in Addison's disease if a crisis is pending, or has appeared, 3 per cent sodium chloride is most beneficial. Also, many surgeons routinely administer 1500-2500 cc. of normal saline as such, or with 5 per cent glucose added, following extensive abdominal operations where there has been considerable handling of viscera.

In cases where both cardiac and renal failure are present, a slow intravenous of 5 per cent glucose in distilled water is safe. Also, this solution is the *only* one that should be used where there has been a recent coronary thrombosis with cardiac infarct. 5-10 per cent glucose in distilled water can be used post-operatively; and following brain surgery where it is desired to lower the intracranial pressure, 50-100 cc. of 50 per cent sucrose (or glucose) in distilled water is used.

To treat rapidly falling blood-pressure post-operatively, either as a result of surgery or anaesthesia, 5 per cent glucose in normal saline is recommended. Gum acacia can also be used; this is excellent in shock from all causes, is used in hemorrhage from ulcers, and is a substitute for blood or concentrated serum. It makes a fine emergency fluid while arrangements are being made for transfusion.

In diabetic coma, 2000-2500 (maximum) cc. of 5 per cent glucose in normal saline, with sufficient insulin to cover the sugar, is practically routine treatment, and prevents the patient from going to the other end of the scale, insulin shock.

Physiological saline, and solutions of 2 parts 5 per cent glucose to 1 part normal saline, are used extensively at the Hospital for Sick Children in children suffering from toxemia as the result of infection, and inanition. Concentrated serum, a product of the Department of Physiology and the Connaught Laboratories, is available for the fighting forces, being inferior to whole blood only, and having the advantage of requiring no cross-agglutination tests. It is unnecessary to state that the best of all fluids is whole blood, correctly typed and cross-agglutinated. It should further be added that "all solutions for intravenous use should be prepared with extreme care so that no reactions will be caused by them. Only fresh solutions should be used, and these must be bacteriologically and chemically pure."[2]

A word about the rate of flow:—there is rarely any reason for flooding fluids into a patient. Prolonged flow of a regulated, small amount is better than a short period of letting the apparatus run wide open. The fastest flow is used in diabetic coma, and sudden shock; in these the rate is 600 cc. the first hour, then 200-300 cc. per hour after this. If the patient is taking little per os, 2500-3000 cc. per 24 hours is plenty. A good average rate is about 40 drops per minute—100-125 cc. per hour. In children, the rate should be 8-15 drops per minute, or 500-700 cc. per 24 hours.

Dangers, or complications:

1. Phlebitis
2. Septicemia
3. Superhydration—cardiac failure, pulmonary oedema

This can be prevented by limiting the saline. It is *most* important to listen frequently to the lung bases posteriorly, and

watch the sacral area carefully for oedema.

Technique

The materials required are a glass funnel (preferably graduated), rubber tubing, drip-bulb, adapter, needles and hypodermic syringe. These are all assembled and put together and sterilized. The funnel is then partly filled with the fluid, which is allowed to fill the tubing and expel all air bubbles. The end of the tube just above the adapter is then clamped, till ready for connection to the needle.

The site chosen will depend on the age and condition of the patient, the fluid to be administered, and the size of his veins. In children, or if a cut-down is required on an adult, the long saphenous vein, just anterior to the internal malleolus, is commonly used. In special cases, (following abdomino-perineal operations) where a cut-down is necessary, an arm vein may be used. The commonest site is any of the veins in the cubital fossa, or the flexor surface of the forearm or wrist, or the dorsum of the hand. If in the region of the cubital fossa, or the patient is delirious or unconscious, it is essential to splint the arm and forearm with an arm-board, to prevent the patient from flexing the elbow and disturbing the needle.

Regarding needles, "in intravenous work more failures are due to dull needles than to any other single factor".[2] Those with a long bevel go into the vein easier, but also on through it and out the other side just as easily. Short bevelled ones are difficult to insert, but once in are much less apt to rend the wall and go out again. The size will naturally depend on the size of the patient's veins, always using the largest needle possible; about 19-gauge is best. For blood transfusion, an 18-gauge, or a cut-down with cannula, are preferable. Gold needles are used at the Hospital for Sick Children, it being considered there is less danger of splitting the vein.

A tourniquet should be applied, just tightly enough to occlude the venous return but not the arterial flow. The patient can help by slowly opening and closing the hand. Also, veins can often be made to stand out by lightly flicking the overlying skin with the fingers. The chosen site is then sterilized with iodine and alcohol, or 65 per cent alcohol alone. With aseptic technique the needle is fitted to the syringe; the skin overlying the chosen vein is drawn slightly to one side, the needle inserted bevel side up, through the skin. The skin is then allowed to roll back over the vein, and the needle is slowly and gradually shoved along the long axis of the vein, almost parallel to the skin surface. As soon as the lumen is reached blood will appear in the barrel. The tourniquet is then loosened, the syringe pulled off the needle, and the adapter (the fluid flowing through it) is attached, one hand all the while holding the needle as stationary as possible. The angle of the needle with the skin providing greatest flow is determined by gently raising or lowering the end of the needle. It is fixed at this angle by wedges of absorbent cotton, and the base of the needle secured by one or two strips of adhesive tape, as is the adapter and the adjacent foot or so of rubber tubing. The arm should be loosely bandaged, and tied to an arm-board, or the wrist to the bed, if necessary. The rate of flow is regulated by the adjustable clamp on the tubing above the drip-bulb.

When cut-downs are required, the technique is as follows: the site chosen for the injection is just anterior to the internal malleolus, over the long saphenous vein. The skin is sterilized and the foot and ankle draped with sterile towels. "The skin across the malleolus is infiltrated with 0.5 per cent solution of novocain. A transverse incision ¾ to 1 inch in length is made across the lower end of the malleolus with two-thirds of the length of the incision anterior to the malleolus. The dissection of the vein is carried out by the separation of the blades of the manicure scissors. The tips of the scissors should be kept in view so as not to cut the vein. The vein can be readily seen and picked up on a strabismus hook. The dissection of the vein is extended by means of the scissors until about ½ inch is freely exposed. A ligature of catgut is now placed about the distal portion of the exposed vein by means of the curved

mosquito forceps and tied. The ends of this ligature are left long so that traction can be exerted on the vein. A second ligature is placed at the proximal portion of the exposed vein and loosely held with a single knot. A slanting transverse nick is made in the vein by means of the 3-inch scissors and the gold needle with the adapter and rubber tubing attached is gently inserted. The reason a gold needle is used is that it is more readily inserted than a steel needle of the same size and does not split the vein so readily on account of the absence of the sharp angles of the steel needle. When the point of the gold needle has been inserted in the vein past the proximal ligature this ligature is tied to hold the needle in place. A syringe containing saline or glucose is attached to the rubber tubing and the injection of fluid started in order to ascertain that the needle is accurately in place and the vein patent. Slight traction is exerted on the vein at this time and during the entire period of the injection by means of the distal ligature. The patient is now ready for the injection of blood".[4]

In Conclusion

In conclusion, let us emphasize one fact: the pendulum has tended to swing too far to the modern routine of large amounts of fluid post-operatively — in many cases too much—in contradistinction to the former custom of too little fluid. Then, too, there are unsuspected lesions — for example mitral stenosis — whose presence is discovered only at autopsy, death resulting from literally "drowning" the patient with fluids.

The author wishes to express his sincere thanks to Dr. John Hepburn for his very helpful and constructive criticism of this article; and to Dr. A. W. Bagnall for his information regarding indications for, and fluids used, and technique of administration.

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"Peace With Honour?"

"Peace, Sir, is a defirable Thing for any Nation, efpecially a trading Nation; but, whoever thinks that a Peace ought to be purchafed at the Expense of the Honour of his Country, will, at laft, find himfelf egregioufly miftaken. By the Cuftom of all Nations, and all Ages, when an apparent, and real Affront has been put upon one Nation by another, the injured Nation ought to make a peremptory Demand of Redrefs, and if it is not granted upon the very firft Demand, they aught to break off all further Correfpondence, and immediately begin Hoftilities. If they fubmit to a Negotiation after such a Refufal, they may, at laft, obtain a Nominal Peace, but it comes attended with Contempt; and a Nation that is contemned, can never expect a Ceffation of Hoftilities."

The Rt. Hon. M. Agrippa.
Journal of the Proceedings and Debates
of the Political Club.
December, 1738.

ROLE OF THE DIPTERA IN BACTERIAL TRANSMISSION

By R. F. WARREN, B.Sc., '44

No other order of insects has so deservedly won the condemnation of medical men generally and perhaps no order has been investigated so thoroughly as the Diptera. This has been done both from the standpoint of their ability to act as agents of bacterial transmission and as hosts to bacteria. It is known for example that some bacteria depend on these diptera in the tropics for a satisfactory completion of their life cycle.

The Diptera is one of the largest orders of the Insecta and includes such common members as true flies of many descriptions, gnats, midges and mosquitos. Over fifty thousand distinct species of diptera have been described and studied. The main characteristics of the order are the mouthparts which are piercing and sucking, or rasping and sucking, the metamorphosis which is complete and the presence of one pair of wings and the vestiges of a second pair as halteres attached to the metathorax.

The diptera are specially adapted to carrying bacteria by reason of specialized mouthparts, methods of feedings, bodily characteristics and habits of living. Their distribution is world-wide and having wings they are able to serve effectively in spreading disease from one neighbourhood to another. Some species are amongst the world's fastest fliers and may rival the Hymenoptera in their range.

A nodding acquaintance with some of the more easily observed features of this order is essential to an understanding of how bacteria can and are transmitted by them to animals and man. These will be briefly discussed under (1) mouthparts, (2) bodily characteristics, (3) metamorphoses and habits of living.

Mouthparts

(*a*) *Blood-sucking type.* While there are a great many different varieties of mouthparts in this order from the more primitive blood sucking type to higher more modified forms, all members can be roughly divided into (1) those forms with piercing apparatus and (2) those forms where this apparatus has been supplanted by rasping and sucking apparatus. The distinction is not a hard and fast one, but serves our purpose here of showing two means by which bacteria are related to anatomy and feeding of insects.

Considering these lower forms, like mosquitos and horseflies, we find a pair of mandibles like sabres and a pair of maxillae ensheathed in a single labium which together with an epipharynx and hypopharynx is prolonged into a tube like projection for sucking liquids. The hypopharynx further has a function of containing the salivary duct which opens on to the most distal part of this tube, and hence it is at the bottom of any incision made by the mandibles.

In the case of the mosquito, an irritating fluid is poured through the duct into the incision. This acts in such a way as to increase the blood flow in this area, which the insect then draws back with some of the saliva into the stomach. This is not accomplished however until such bacteria harboured in the saliva have had a chance to escape into the tissues and blood. They are deposited hypodermically so to speak in the new host. Many are withdrawn with the blood but there is no guarantee that this is complete, and it is known such diseases as malaria obtain their entry into the human host in exactly this manner.

By this means diptera of the blood-sucking type can transfer bacteria living in the blood from one case to another providing the bacteria can survive the interval between the insect's meals and other conditions within the insect's body.

Mosquitos have to their credit the satisfactory transmission of malaria

already mentioned, yellow fever, filariasis, elephantiasis, dengue and many other diseases which affect animals.

Nor is the mosquito alone in this respect. Glossina palpalis better known as the tse-tse fly is responsible for the transfer of sleeping sickness and nagana. In this case it has been suggested that some other host besides man is susceptable. Depopulation of a district has been shown to be ineffective in stamping out the disease for the trypanosomes have been demonstrated two years after depopulation, an interval greatly exceeding the life span of the agents of transmission. Antelopes have been suspected of playing a role in the trypanosomes' cycle, but this has not yet been settled. Koch claimed that crocodiles played a similar part here and advocated the destruction of crocodile eggs in this regard.

Stomoxys calcitrans, an insect like our common stable fly is supposed to be implicated in a similar manner in carrying surra (Trypanosoma evansi).

In Canada a plague like disease of rabbits is not uncommonly conveyed to man by the bites of the Simuliidae but unlike the disease in rabbits produces only a mild fever with low death rate. Tularaemia as it is called enters the skin in the manner already described for mosquitos. The bites occur around the neck and especially behind the ears in late summer. The insects responsible are known to some as "Black flies" or "Sand flies".

(*b*) *Non-blood-sucking type.* This is a higher form of the diptera who are unable actually to pierce the skin because the mandibles and maxillae if evident at all are only present as vestiges while the labium has degenerated so that the palps have become merely lobules of the organ. Nevertheless, their methods of obtaining food, though entirely different to those already considered, are just as capable of causing bacterial infection.

The food is made liquid by a process of rasping and by the habit of regurgitating gastric contents to lubricate the process. When sufficiently acted upon in this manner the whole, or part of it, is sucked up but not before any bacteria in the insect vomitus has had a chance to be transferred to insect food. Accordingly these types of diptera carry those bacteria which invade man with his food, into the alimentary tract, whereas the former blood-sucking group are more adapted for transmission of haemophilic bacteria.

The worst offender is the housefly, Musca domestica, who is largely responsible for typhoid carrying, though their methods of eating lend themselves admirably for the propagation of cholera, dysentery, infantile diarrhoeas, and tropical ophthalmia.

Bodily Characteristics

Here again the Diptera have shown themselves excellently provided for picking up and depositing bacteria and the things bacteria thrive in. All insects are covered by a chitinous exoskeleton but the strength and thickness of this protective outer coat varies from order to order. In the Coleoptera for example this armour reaches its heights in brilliancy and strength. But in the Diptera it is thin and soft and the surface is covered with hairs, especially over the thorax and legs. These retain all manner of infested filth whether decaying matter, faeces or only water, from which the insect may have hatched. It is the fact that these contaminated places are so frequently the breeding places of insects that make them of even greater importance medically.

Another bodily detail which aids bacteria is the number of soft sticky pads found on the feet of flies. By means of these they are able to maintain an inverted position from a perfectly smooth surface but by these same pads they are able to hold firmly bacteria which become scattered wherever the fly walks. One has only to observe an agar plate after a fly walks over it to see this fact well illustrated.

Habits and Metamorphoses

The habits connected with feeding have already been mentioned.

The habit of houseflies indulging in frequent manicuring operations on the sugar bowl has been seen by everyone.

Perhaps their equally frequent habits of defaecation have not. All diptera have a complete metamorphoses. In some this has been modified to the egg remaining within the female until hatched and the larvae subsequently born alive.

The bacteria offenders have not remained so discreet and in the housefly the eggs are laid in bunches of about one hundred and twenty-five in fresh horse manure, so that upon hatching the larva merely burrows until after fourteen days it penetrates deeper to attain the pupa stage. After the second transition the adult reaches the surface by a curious process of blowing up a balloon from the head end, pushing the surrounding matter aside and crawling into the prepared space to repeat the process.

Mosquitos prefer water and make their transition in that medium but the bacterial contacts and contaminations come when they begin to feed.

Not a few of the diptera lay eggs on living matter and the larva burrows into its erstwhile host to obtain nourishment. In tropical countries varieties such as the Tumbu fly, Corylobia anthropophaga, also known as the Ver du Cayor, lay their eggs on the heads and necks of small children and young dogs. The breaking of the larvae into the skin is painless and unnoticed. During the ensuing twelve to fourteen days the larvae may be found at the bottom of a boil-like eruption with hollow centre. This pustule may and does become the centre of secondary bacterial infection for which the insect is indirectly responsible. As many as fifteen and twenty of these large larvae which measure up to half an inch in length have been recorded as removed from the head of a single child.

Summary

Diptera have been considered in the part they play in aiding and transmitting bacteria to humans. The mouth-parts of both blood and non-blood-sucking types have been described from their ability to assist bacterial entry. The bodily characteristics and habits of growth of the order as a whole have been seen to be of the utmost importance in bacteria transmission. Further, the metamorphoses of diptera and the parasitic nature of certain members show the order, as a whole, to be of great medical interest.

The author wishes to thank Dr. Price for reading the paper and offering suggestions.

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Poem

Diogenes, the famous cynick,
Who did all men and manners mimick,
As he was with another walking
E'fpy'd two women clofe a talking:
Beholde! faid he, (he wou'd not Ken 'em)
The afp and viper changing venom.

Trionymus

Alumni Notes

By K. C. GREER, M.D.

The majority of the well-planned internships which we anticipated a year ago are progressing according to schedule but already the wheels of fate are at work, as evidenced by the following items of news.

* * *

Crawford Shier, at a quiet wedding on November 28, was married to Miss Frances Page. Judging by the duration of the courtship there should be no doubt in the groom's mind that he had made a perfect choice—a certainty of which we have been aware for some time. The bride will reside on South Drive—her husband will no doubt lead "Locating at the General" a merry chase.

* * *

Taylor Statten has followed suit. He rook the plunge into marital bliss on December 7. The happy bride is Miss Al. Turner—another childhood romance with a happy ending. We thought the stag party for Crawf. was an outstanding success, but we bow our heads in respect for the instigators of an affair which took place recently in a down-town hotel.

* * *

Alfred Crossland is still one jump ahead of our two newest nuptial adventurers and is now busy raising a family. Our only indication that the blessed event is not far off is that on his frequent visits home Alf. is never without his stethoscope.

* * *

George Manning, late of the intern staff of St. Michael's Hospital, has joined the Air Force and is now known as an F.O. At present he is stationed at the Banting Institute, engaged in research in Aviation Medicine.

* * *

Norman Terwillegar left for active service with the Navy on December 2. At a farewell party at the Western Hospital on the preceding evening his knowledge of naval matters was not astounding, but he did look impressive in a surgeon-lieutenant's uniform!

* * *

Roderick Ross was scheduled to leave for active service with Twig, but a few days prior to his departure he developed an illness which as yet has not been diagnosed with certainty and so he remains on the Naval Reserve.

* * *

Lemuel Prowse left St. Michael's during the summer for army training in Prince Edward Island but a recent illness has necessitated a sick leave at home.

* * *

Robert Northover has recently left the intern staff of the Western Hospital to take an internship at the Toronto Military Hospital with a salary which should allow him to join the ranks of our married men when he chooses (if the male does the choosing).

* * *

Reginald Allen's internship at St. Michael's Hospital was a short one and he is now making fame and fortune in Canada's northlands. The address is Gunnar Mines, Manitoba.

* * *

We wish to congratulate the editorial staff of Volume XVIII on their first issue and to thank them for their co-operation in the publication of alumni notes.

Abstracts

R. L. MacMILLAN, Editor

Contributors J. G. Watt, D. J. Breithaupt, D. P. Bryce, R. W. Pritchard, W. E. Armour

CARCINOMA OF THE VULVA

By W. G. COSBIE, Toronto
C.M.A.J., Nov., 1940

The author's study is based on fifty-nine patients who have been treated for carcinoma of the vulva in the Gynaecological Department of the Toronto General Hospital and the Ontario Institute of Radio-Therapy, Toronto, since 1929. Fifty-six had squamous cell carcinoma, two melanotic sarcoma, and one carcinoma of Bartholin's gland.

Carcinoma of the vulva is a disease of later life, most cases being over sixty years of age, although it occurred in a few cases between forty and sixty years of age.

Pruritis vulvae is the commonest symptom. Pain occurs with large tumours in the vulva. Often there is no discomfort and the patient complains only of swelling. Dysuria and frequency are common complaints with a tumour in the vestibule or with an ulcerated growth, irritated by urine.

The commonest situation is the labium majus. The growth usually appears as a surface plaque or as superficial ulceration. Next situation is in the labium minus, originating in the prepuce and involving the clitoris. A few originate in the perineum, and a few in the vestibule. The tumour of Bartholin's gland appeared as a hard nodular swelling beneath the epithelium.

Advanced tumours markedly destroy tissue and ulcerate into the lower vagina. The inguinal nodes are frequently enlarged.

Twenty-one cases had leukoplakia vulvae. All of these suffered from pruritis or pain of the vulva.

The symptoms in nearly all cases were present for over a year. Nine had five years of symptoms. Medical advice was neglected due to fear or modesty. Malignancy is low grade but insidious.

Vulvectomy is the treatment of choice (Taussig). Those treated by irradiation later had to have vulvectomy due to the unhealthy state of the skin.

With radical excision of the vulva and the inguinal lymph glands the five-year cure rate is sixty-five per cent. It is only three to six per cent with simple vulvectomy.

The technique of the operation is described.

J. G. W.

RHEUMATIC HEART DISEASE

PATHOGENESIS AND ETIOLOGY IN THEIR RELATION TO THERAPY AND PROPHYLAXIS

By HOMER F. SWIFT, M.D.,
From the Hospital of the Rockefeller Institute for Medical Research
Medicine **19**, *417 September 1940*

This is a lecture presented to the New York University College of Medicine in January, 1940, filling 21 pages, illustrated by pathological pictures, and is perhaps rather rambling.

Pathologically, the noxious agent of rheumatic fever is seen to affect primarily the supporting structures of the body, viz., the collagen and elastica, varying from a swelling of the ground substance between the fibrils to actual necrosis of the fibres. The inflammatory response to this is peculiar in that there may be excessive oedema or exudate, and that this exudate disappears rapidly under adequate dosage of certain antipyretic drugs. The exudative characteristics may be absent later in the disease when

proliferative lesions indicate continuing or renewed injury to the tissues, as though enough immunity were set up to lessen the severity of the tissue reaction, but not to obviate it completely. Examples of this are the subcutaneous nodules and small areas of pleurisy and pericarditis which may appear even when the patient is receiving large doses of antirheumatic drugs.

The rheumatic granuloma cells are gradually transformed into fibroblasts, and scar tissue, resulting in more collagen which is especially vulnerable to the "rheumatic poison." Motion and functional strain are considered important in the localization of the rheumatic injury, and for this reason the heart and blood vessels seem to be peculiarly vulnerable. Extensive lesions may appear within two to three weeks of the attack of rheumatic fever. It would seem that all the valve rings are involved, but verruca (wart) formation and permanent thickening are limited to the aortic and mitral valves, due to the greater functional trauma to which they are subject. "In the following order: mitral, aortic, tricuspid, and pulmonic valves, respectively, are the stress and strain to which they are submitted progressively reduced; and in the same order are they the bearers of verrucae and the sites of scarring deformities." (Why he should choose this order of "functional trauma" is not evident.) In support of the increased work theory he quotes experiments by Pearce in rabbits infected intravenously or intratesticularly with virus III. Without trauma only small cardiac lesions were induced. Functional trauma following the injection of gum acacia solutions resulted in a relatively large number of right heart lesions; pitressin injections were followed by relatively more left heart lesions; and cardiac punctures resulted in the largest proportion of pericarditis cases. Because of the reduced pulse rate obtained by antirheumatic drugs, the resultant rest to the heart is considered to be very influential in saving the heart a material amount of damage. Other experiments showing the effect of work on an injured organ are quoted, e.g., experiments in which nephritis caused in rats by a specific nephrotoxic serum, is maintained severe and fatal, or diminished, according to the high or low protein diet fed the animal.

Rheumatic fever he considers to be in some way related to a streptococcus of the Lancefield group A, but the exact relation is unknown. Precipitins developed against "M" substance, a type specific component, were measured in patients infected with haemolytic streptococci. Most non-rheumatic patients developed strong anti-M precipitins by the fourth or fifth week; the rheumatic cases with short courses developed high titres early, while those with long subacute cases of rheumatic fever did not develop high titres until late in the disease. This suggests that possibly the long persistence of the disease is related to low type specific immunity against the haemolytic streptococci.

Sulphanilamide, in doses of two to three grams per day, given to rheumatic children in the quiescent stage of the disease, was found to reduce the incidence of rheumatic relapses from 20 per cent in controls, to 1 in 184 patients studied, during the course of one year.

It is supposed that partial but not complete immunity of the rheumatic patient to haemolytic streptococci would explain how the organisms retain only a low grade irritative capacity in the tissues; and a reactive state in the tissues called hypergy would account for the manner in which the tissues respond to a very small stimulus. Post mortem cultures of group A haemolytic streptococci are reported by Green and Collis in a large number of cases, from the heart valves or pericardium, and even peritonsillar tissues, mediastinal lymph nodes and spleen. The protection of patients from repeated haemolytic streptococcal infection is seen to offer some hope of preventing recurrences of rheumatic fever, and prophylactic measures to this end are especially important in those who have previously suffered from this disease.

D. J. B.

CANCER OF THE RECTUM

By J. A. MacFARLANE, F.R.C.S., (EDIN.), LT.-COL. R.C.A.M.C., Toronto
C.M.A.J.

Cases of rectal cancer are being sent to hospital for surgical treatment more often and in an earlier stage than twenty years ago, due to improved methods of diagnosis, and to the work of Miles, Lockhart-Mummery, Jones and Devine who have successfully perfected surgical techniques that have a reasonably low mortality rate.

Diagnosis: *Symptoms*—All complain of bleeding early in the disease. This may be mistaken as due to haemorrhoids.

Signs—Every case of rectal carcinoma can be diagnosed in the examining room either by digital palpation or by proctoscope. Radiology is not very helpful.

Operability: Between 1930 and 1940, sixty-three per cent of the cases admitted to the Toronto General Hospital were operable. With suitable preparation some cases that have fixation of the growth to the prostate or sacrum are operable. If the tumour has extended through the pelvic floor and there are peritoneal implants it is useless to operate. This also applies to secondaries in the liver. Rectal carcinoma is comparatively slow growing.

The operation should be chosen with care and the procedure used that is best suited to each case.

The one stage abdomino-perineal excision of Miles is radical but good. It cannot be used if the lower bowel is found to be insufficiently clear when the abdomen is opened.

In such a case the two stage operation of Lahey is preferable. First is division of the colon. Then 10 to 14 days of washing of the distal loop. Resection of the bowel is performed when the distal loop is clear.

In very old people and those who for various reasons are considered poor risks, a preliminary colostomy followed in two weeks by perineal resection is the method of choice.

Turning in of the lower segment of the rectum following a preliminary colostomy and intra-abdominal resection is indicated when the growth is higher in older individuals.

Devine safely removes growths with retention of the anal sphincter when the tumour is recto-sigmoid. This can be used where a Mickulicz is impossible for lack of mobility. The glands in the mesorectum and hollow of the sacrum must still be cleared out.

Radiotherapy has a very limited use.

Twenty-four per cent were alive and well five years later. Thirty-nine per cent were alive and well for a period ranging from one to five years.

The general operative mortality is seven per cent. Improved technique will doubtless lower this figure.

J. G. W.

A CLINICAL STUDY OF TETANUS

By RALPH SPAETH, M.D.
From the Department of Paediatrics, College of Medicine, State University of Iowa
American Journal of Diseases of Children **60** *130, July 1940*

This interesting 30-page monograph concerns itself chiefly with the treatment of tetanus, with which it deals in specific detail, as well as also considering the value of surgery, antitoxin, diagnosis, prognosis and prophylaxis. Dr. Spaeth has apparently had considerable experience with this rather rare condition, and reviews 66 cases, 33 before, and 33 after the inauguration of the regime he proposes. In considering the results, 9 of the 33 cases since 1932 died, whereas 12 of the preceding 33 died. When the "mild" cases are excluded from this series, the results are more dramatic, as the later cases were apparently more serious than the earlier ones. Ten of sixteen died before 1932, and nine of twenty-six since, a difference in case fatality rate of 62 per cent to 34 per cent.

The primary requisites of his treatment are sedation and antitoxin. As sedatives, he recommends avertin with amylene hydrate, and/or sodium amytal. He also advocated printed routine instructions and careful observation of each patient. The indications and

dosages for both drugs are meticulously pointed out.

Serum therapy is considered useful enough to be given routinely to all patients on admission (routinely 30 minutes after injection of epinephrine and atropine, dosage graded according to age, to minimize reactions) in the form of a single injection of 30 to 60 thousand units. (Tetanus toxin has been proven to travel in the blood stream instead of by way of the peripheral nerves as was formerly taught.) Antitoxin is given intravenously if at all possible, or intramuscularly, and the intrathecal route is condemned. The treatment of immediate and delayed serum reactions is discussed.

Surgery is not considered to be an emergency procedure, but should only be done after sedation has been accomplished and antitoxin administered.

The most dreaded complication was pneumonia, 14 of the 17 patients who had pneumonia died—21 cases were fatal.

Diagnostically the clear mind, the absence of fever in uncomplicated cases, and the occasional absence of trismus is stressed.

Prophylaxis is discussed, and the author recommends 5,000 units of A.T.S. in any contaminated injuries, up to 20,000 units if the patient is not seen until the day after the injury, and there is reason to believe the wound may be infected. He considers that tetanus toxoid should be combined with diphtheria toxoid, and given to all children, in doses of 1 cc. at intervals of 2-3 months, for 2-3 doses. (The Connaught Laboratories recommend 1, 1.5, and 1.5 cc. doses at 4 week intervals, plus 1.5 cc. every 2 years and on injury.) At the time of injury a further injection of 1 cc. of toxoid is given to augment the blood antitoxin level. This is said to give a protective level for about 7 days following the injury, and the usefulness of the first injections is said to last for several years. The combined diphtheria-tetanus toxoid gives no more reactions than diphtheria toxoid alone, and is said to cause a better response than if they were administered separately!

This article is an excellent reference for the therapy of an acute severe disease, rarely met in practice, but one in which the doctor is likely to be successful if he can only tide the patient over the acute stage.

D. J. B.

VACCINATION AGAINST TUBERCULOSIS WITH BCG VACCINE

J. A. BAUDOUIN, M.D.
Director, School of Applied Social Hygiene, Faculty of Medicine, University of Montreal

Canadian Public Health Journal, **31**, *362, August 1940*

This article presents a concise summary of the work carried out by Dr. Baudouin in conjunction with the Institut Pasteur, Paris, and the National Research Council, Ottawa. The work commenced in 1926, since which time, in 13 years, there have been some 20,000 vaccinations with the modified tubercle bacillus of Calmette and Guérin as prepared by Dr. Frappier, the Professor of Bacteriology of Montreal University.

This paper concerns itself with those children in contact with positive-sputum cases of tuberculosis. Of these there were 725 vaccinated, and 1,178 unvaccinated, serving as controls. All vaccinations were done within 10 days of birth, and were apparently harmless as some 9,000 were done without untoward reactions, and the vaccinated group living in contaminated environment showed a mean reduction in general mortality of 50 per cent.

The tuberculosis mortality was 11/725 in the vaccinated group and 58/1178 in the controls, or roughly 1.5 per cent and 4.8 per cent. Unfortunately, the exact figures for his comparisons of morbidity and duration of immunity are not given, but merely the percentage of one compared to the other. These throughout are favourable to the vaccinated group, but in a small number of cases a very high percentage readily accumulates statistically, with little real meaning. The author is very well aware of the value further cases will add to the study.

The following are the conclusions which he considers may safely be drawn. Vaccination by Calmette's method does increase human resistance to tuberculosis, which lasts for 2 or more years. Vaccination of new-born babies with BCG vaccine should be included in any campaign directed against tuberculosis. All babies born in families in which a patient is suffering from tuberculosis should be vaccinated. When the baby cannot be removed immediately after birth to a tuberculosis-free environment, the best possible protection which can be offered against early infection, with its most dangerous consequences, is vaccination. BCG vaccine can be safely administered to all new-born babies because the procedure has been proved to be absolutely harmless.

There are others who think that for most people a negative tuberculin test is desirable as long as possible, and that an allergic response is developed to the toxins of tubercle bacillus by early infection or vaccination. However it is usually conceded that in a case where the child cannot be removed from active tuberculosis, vaccination is useful.

D. J. B.

SYMPOSIUM ON THE OESOPHAGUS

Arch. of Surg. Vol. 41, No. 5

This issue of the *Archives of Surgery* has to do mainly with a collection of modern views and experiences, with regards to surgery relating to the oesophagus. Obviously in this abstract we can merely indicate briefly the subjects dealt with in the hope of guiding those whose interest leads them to the perusal of this work. The papers included are:

1. Diseases of the Oesophagus by L. Clerf

This is a general clinical review of diseases of the oesophagus with emphasis on treatment and the use of an oesophagoscope.

2. Atresia of the Oesophagus by Thomas Larrman

The embryological factors are briefly reviewed and certain conclusions are drawn as to the most advantageous surgical treatment. Hope of successful surgical measures lies in early operation and improvement of technique to allow direct anastomoses in as many cases as possible.

3. Roentgen Demonstration of Oesophageal Varices by Richard Schatzki

In this paper the author discusses the roentgen diagnosis of varices and points out the significance of such procedures in the early recognition of cirrhosis of the liver and primary carcinoma of the liver as well as the differential diagnosis of hematemesis, ascites and splenomegaly.

4. Bleeding Oesophageal Varices by Walters, Moersch, McKinnon

Etiological factors are reviewed and the modern therapeutic measures such as splenectomy, ligation of the coronary vein, omentoplexy and the oesophagoscopical injection of sclerosing fluids are discussed.

5. Oesophageal Diverticula by Lahey

The author reviews the history and pathogenesis of the different types with particular emphasis on the problem of the pharyngosophageal diverticulum.

6. Conservative Treatment of Achalasia by E. B. Freeman

It is concluded that the Plummer air or water dilator is the best instrument available to afford complete dilatation of the cardia and that such a procedure should be considered before surgical intervention is attempted.

7. Surgical Considerations in Achalasis by O. Ochsner, M deBahey

Here is a review of the etiology followed by a classification of the radical treatment into that directed at:—

(1) the oesophagus,
(2) the cardia,
(3) the diaphragm,
(4) the nerve supply.

It is concluded that oesophagogastrostomy is the most successful procedure and as such is outlined most fully.

8. Surgical Treatment of Carcinoma of the Oesophagus by John Garlock

The incidence, diagnosis and prognosis are discussed. Cures are classified as to position and a detailed discussion of the surgery is given. D. P. B.

INSTABILITY OF TUBERCULIN REACTIONS

By A. W. COHLSTROM
Amer. Rev. of Tub., Oct. 1940

Examination of dispensary records of almost 40,000 members of over 500 families under observation for periods of five to fifteen years indicated that 11 per cent of positive reactors became negative during the period and that the reversal of the reaction varied inversely with the original degree of sensitiveness. The overwhelming majority of unstable reactions were children.

R. W. P.

LEUCOCYTE COUNTS IN TUBERCULOSIS

By E. M. MEDLAR
Amer. Rev. of Tub., Oct. 1940

The author regards the relatively simple leucocyte index, which takes into account the four essential variables, viz., lymphocytes, neutrophiles, monocytes and the total leucocyte count, as being most useful in tuberculosis. It is of value in certain sub-clinical cases: in the determination of activity and the likelihood of relapse. In the interpretation of the index the leucocyte picture of a normal healthy adult is used as a yardstick.

R. W. P.

THE DETERMINATION OF PHYSICAL FITNESS

By H. H. KESSLER
J.A.M.A., Nov. 9, 1940

This is a subject of great current interest, and as such is dealt with in a broad common sense manner. The difficulty of application of the principles outlined is not discussed.

Physical fitness is defined as a socio-economic concept, involving the social evaluation of a series of anthropologic, physiologic, and psychologic traits. It is claimed that the physician also derives a social judgement from pathology.

In its widest sense physical fitness is the ability to perform productive and continuous work, which is based on native ability, training and the presence or absence of illness.

It is stressed that in the evaluation of this condition physical examination, in the absence of active disease, is of little value and that the presence of static defects is of little significance due to the factors of training and compensation.

Also the author stresses the fact that after all the range of human capacity is small and that the average job can be handled by the average man.

D. P. B.

PREVENTION OF DIABETES

R. E. HAIST, J. CAMPBELL, C. H. BEST
New England Journ. of Med., Oct. 17, 1940

If a very large portion of the pancreas is removed in animals, the animal becomes diabetic. Examination of the remnant shows progressive degranulation and finally hydropic degeneration of the islet cells. Apparently the cells become sick and then die from exhaustion. The injection of Ant. Pit. extract causes a similar exhaustion. It does this by increasing the demand for insulin. The diabetic condition brought on by overstrain in the above two ways can be prevented by reducing the demand for insulin. This may be done by fasting, by a high fat diet or by giving insulin. It was found that the insulin content of the pancreas of normal rats fed on a fat diet was much lower than if fed on a carbohydrate diet. The fat diet, however, did not cause degeneration of the cells of the pancreas. Hypophysectomy had no effect on the above findings.

Factors that prevent disease should also limit its progress. The authors believe that their results should be of value. Fat feeding, fasting and insulin administration prevent the development of diabetes in animals presumably by decreasing the demand for insulin and hence preventing an exhaustive irrversible degeneration of the islet cells. These factors rest the pancreas and there is no evidence that resting the

pancreas causes degeneration. In fact regeneration and restoration of the islets may take place. This treatment should be best suited to an early stage of the disease. The authors outline an experiment to be carried out on children that should yield valuable information.

An important observation is that the administration of Ant. Pit. extract causes a proliferation, with mitotic figures, of the islets as well as degeneration of the islets. When insulin is given with the extract there is still proliferation but there is not any degeneration. This combined treatment may be of value in stimulating the islets in human diabetes. It is not mentioned in the article whether or not, in the ordinary case of diabetes mellitus in man, there is exhaustion of the islets as evidenced by hydropic degeneration. If so, the results of the above investigators should be directly applicable to man.

R. L. M.

VITAMINS IN PEPTIC ULCER

H. FIELD, W. D. ROBINSON, D. MELNICH

Ann. of Int. Med., Oct., 1940

On the basis of clinical investigations by themselves and others, these writers have concluded that vitamin C deficiency is important in the majority of cases of peptic ulcer. Some cases of hemorrhage from ulcers may be scorbutic in type; many of such patients were found to have low blood levels of ascorbic acid. Lack of vitamin C may retard the healing of an ulcer.

A diet low in meat and vegetables and continued alkili administration may cause a nicotinic acid deficiency with pellagrous changes.

W. E. A.

TWO FORMS OF GENITAL INFECTION

LISTER AND LEES

B.M.J., Oct. 12, 1940

This article calls attention to the incidence of Trichomonas Vaginalis in males. It is present in 4 per cent of all their cases of venereal disease, and accounts for 16 per cent of all non-gonorrheal urethritis. It is suggested that it be sought for routinely in men.

Leukorrhea, in forty-nine out of two hundred consecutive cases, was found to be due to thrush. The danger of transmission to the new-born child was pointed out.

W. E. A.

ESSENTIAL PROBLEMS OF THE SURGICAL TREATMENT OF INGUINAL HERNIA

L. M. ZIMMERMAN

Surg. Gyn. & Obs., Nov., 1940

The repair of hernia since the time of Bassini has become a routine. The results, especially for direct, are very bad. Gallie states that 40 to 50 per cent of the latter have a recurrence. The underlying anatomy and pathology have been neglected. The author gives a clear, concise, illustrated description of the inguinal region.

He reaches two important conclusions:—

(1) Indirect hernias are due to the presence of a congenital processus vaginalis.

(2) Direct hernias are due to a weakness of the posterior wall of the canal. This is usually a congenital absence of the lower fibres of the internal oblique muscle.

On this basis, repair of an indirect hernia is an entirely separate problem from the repair of a direct one. For indirect herniae in children all that is required is the removal of the congenital sac. Adults, where there is secondary stretching of the internal ring, it may be pulled together without tension by a few fine silk sutures. It is pointed out that any procedure such as stitching fleshy fibres of the int. oblique to the inguinal ligament (Bassini) are entirely without logic and in fact are dangerous in that they are liable to weaken the posterior wall of the canal and render more likely a direct hernia.

For direct hernia the sac is freed from

the stretched fascia transversalis layer and is returned intact to the abdomen. The transversalis layer is stitched together and reinforced with a fascial flap possessing a blood supply. There should be no tension on the fascial flap and no fat or muscle interposed. An excellent description of such a procedure is given. This article is well worth reading and seems to pigeonhole the controversial question of treatment of inguinal herniae.

R. L. M.

THE DIAGNOSIS OF TUBERCULOSIS

H. M. KINGHORN

J.A.M.A., Nov. 9, 1940

The early diagnosis of tuberculosis, especially pulmonary, is one of the main factors that contribute to its curability. The author presents convincing evidence that X-ray examination is by far the most important means of diagnosis at our disposal. For example, of 1,367 consecutive patients diagnosed as active pulmonary tuberculoses, 32 per cent had no physical signs. Diagnosis was made by X-ray.

Another interesting fact was that 40 per cent of patients have cavities shown by X-ray and of these only 5 per cent have physical signs of cavity.

The X-ray appearance of early tuberculosis is described. Much stress is laid in the finding of a mottling with ill-defined margins that blends gradually with surrounding lung tissue. The X-ray appearance correlates with the clinical course to a considerable extent. Active disease is characterized by the ill-defined mottling, while diminished activity is shown by denser, sharply defined areas. The clinical progress can be gauged very accurately by following a series of X-rays taken at intervals of six weeks.

An interesting observation is that, if an individual, reaches twenty-five years of age and his lungs do not show evidence of tuberculosis, it is extremely unlikely that he will ever contract the disease. The author believes that all children should have chest X-rays taken. R. L. M.

TREATMENT OF PULMONARY TUBERCULOSIS

R. C. MATSON

J.A.M.A., Nov. 9, 1940

No specific has yet been found for tuberculosis. Modern progress in the treatment is due to the addition of collapse therapy to the triad of general rest, fresh air and wholesome food.

Collapse therapy hurries up the cure and forestalls the extension of the disease to the larynx and elsewhere in the body. The various methods to produce collapse therapy are discussed and commented upon. Artificial pneumothorax is only applicable to about 40 per cent of cases because of adhesions. Oleothorax is recommended for the treatment of tuberculous pyothorax.

An inadequate pneumothorax may be converted into an adequate one in 20 per cent of cases by the use of a high frequency current cautery and the thoracoscope. This is known as intrapleural pneumonolysis and is a procedure that can be done with the minimum of shock and pain to the patient.

Interruption of the phrenic nerve is now limited to lesions confined to the lower lobe. Here it is combined with Pneumoperitoneum. Repeated crushing of the nerve rather than complete avulsion is desirable.

Thoracoplasty should be adapted in type and extent to the character of lesion. About 60 per cent of patients are greatly improved. It is usually called in when other collapse therapy does not work.

The upper phase thoracoplasty combined with an extrafascial apicolysis (Semb's thoracoplasty) is denounced on grounds that just as good results can be obtained with extrapleural pneumothorax for comparable lesions with a great deal less shock and danger to the patient. The author has devised a method of extrapleural pack which obviates the difficulty in maintaining an extrapleural pneumothorax.

R. L. M.